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# Diseases of POULTRY

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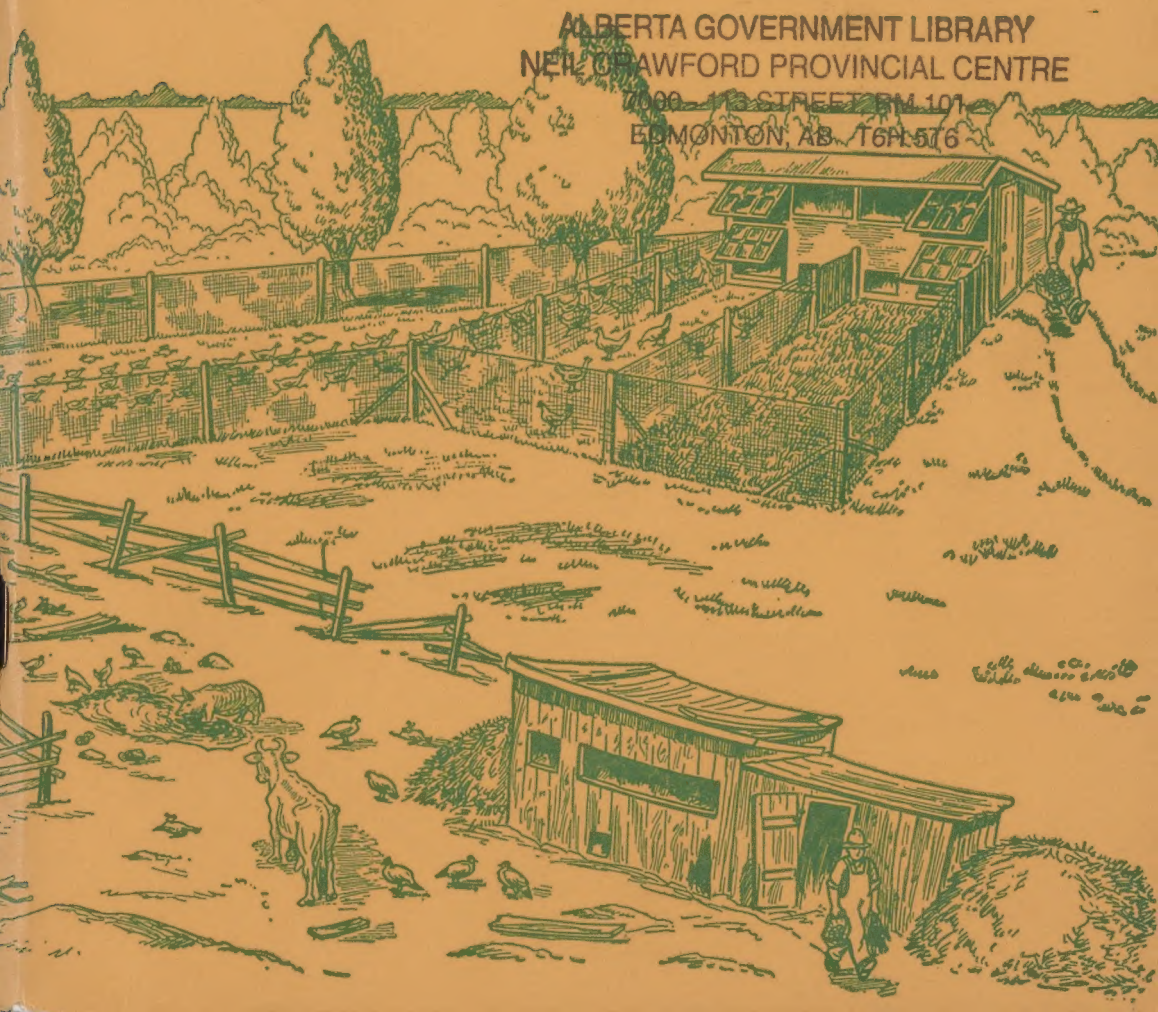

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**UNIVERSITY OF ALBERTA**
**FACULTY OF AGRICULTURE**

# Diseases of Poultry

BY

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COVER—SANITATION PAYS DIVIDENDS
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# Diseases of Poultry

BY

D. R. CLANDININ and A. R. ROBBLEE

## INTRODUCTION

This bulletin has been revised and extended in response to many requests from poultry keepers for information regarding poultry diseases. In the discussion, an effort is made, first, to outline practices applicable to the prevention of disease in poultry, and second, to deal with the more important diseases to which poultry are susceptible.

## PREVENTION OF DISEASE

Domestic poultry are subject to a large number and variety of diseases, some of them highly contagious, many of them incurable, and all of them a source of loss to the poultryman. While treatment may be applied successfully in a limited number of cases, it should not be considered the solution to the disease problem. The most economical and satisfactory line of attack in combating disease lies in prevention. This involves the intelligent application of the principles of breeding, feeding and sanitation, combined with prompt recognition of abnormal conditions, as they occur, and careful, thorough and efficient treatment at the first sign of trouble.

### Breeding.

Of fundamental importance to the well-being of the flock are the inherited qualities of the birds in it. For this reason, the culling, selection and breeding in the flock should be such that constitutional vigour and resistance to disease will be developed. From the time chicks hatch, a rigid and continuous culling of weaklings should be practised. Any birds that appear to be lacking in vigour, unthrifty or diseased, should be culled and disposed of as soon as noticed. Vigorous, healthy two-year, or older birds should be given preference as breeders.

When it is found necessary to introduce new blood into a flock, care should be taken to see that the imported stock comes from sources known to be high in stamina and free from disease. This is advisable, first, because resistance or susceptibility to disease may be transmitted and, second, because many poultry diseases can be introduced into a flock by "carrier" birds.

**Feeding.**

Good feeding has also been shown to play an important role in resistance to disease. Over a period of years, the approximate protein, carbohydrate, fat, fibre, mineral and vitamin requirements of various classes of poultry have been determined. Accordingly, lowered resistance to, and mortality from disease caused by dietary deficiencies need no longer occur, even where birds are kept in strict confinement. The inclusion of these nutrients in the diet in proper amounts and proportions will be found of inestimable value in safe-guarding the health of the flock.

**Sanitation.**

Agents responsible for disease may be introduced into a flock by means of new birds brought in from other flocks; by birds returned from poultry shows; by contamination from feed sacks or shipping crates; by the feet of humans; by wild birds and animals; and by parasites (such as mites). It is wise, therefore, to take precautions against such possible sources of infection by purchasing new stock only from flocks known to be relatively free from disease; by isolating newly purchased stock and birds returned from poultry shows for two to three weeks before placing them with the rest of the flock; by excluding poultry buyers and visitors from the poultry yards and houses; and by providing every possible means of guarding the flock against infection from outside sources.

On the farm, an intelligent and conscientious program of sanitation should be followed so that the birds may live in comfortable, clean and uncontaminated surroundings, and so that in case of an outbreak of disease, its spread can be checked quickly.

The laying house should be located on well-drained land, face south or east and should be protected by a windbreak. It should be of ample size for the number of birds being kept, properly insulated against extremes of temperature, well ventilated but free from drafts, dry and sunny. The walls of the house should be of tight boarding, the floor of cement or good lumber. The inside equipment should include: removable dropping boards or dropping pits, roosts, broody coops, nests, mash hoppers and waterers on stands, grain troughs, and oyster shell, grit and bonemeal feeders. (Plans for the construction of a laying house and poultry equipment may be obtained from the Department of Animal Science, University of Alberta, Edmonton.)

Prior to placing each year's pullets in the laying house for the winter, it should be given a thorough cleaning. Roosts and dropping boards or dropping pits, nests and other equipment should be re-

moved for this purpose. These, as well as the floor of the house, should be scraped, swept, washed with water, scrubbed with lye-water (1 pound of lye to 5 gallons of water), allowed to soak overnight, washed again with water and then disinfected by spraying with a good disinfectant. If mites have been a source of trouble during the previous year, the house and equipment should be treated as recommended under Mites (see Common Chicken Mite). Following cleaning and disinfecting, the walls and ceiling of the house should be given a good coat of whitewash. Whitewash may be prepared by slaking one bushel of quicklime in twelve gallons of water, dissolving two pounds of common salt and one pound of zinc sulphate in two gallons of boiling water, pouring this mixture into the slaked lime, adding two gallons of skim milk and mixing thoroughly.

The floor of the laying house should be kept well covered with clean dry litter. As a general rule the litter should be removed at weekly intervals during the winter, while removal every two to four weeks will usually suffice in summer. Where the deep-litter system of litter management is followed yearly removal of the litter is satisfactory. Nests should be kept well furnished with clean straw. Replenishing nesting material as required and cleaning nests at two-week intervals is advisable. Dropping boards should be kept clean; daily cleaning is recommended. Where dropping pits are used, removal of the droppings at monthly intervals is satisfactory. All material removed from the house should be disposed of in such a manner that the birds will not have access to it.

In the daily routine nothing is more important than the providing of clean, fresh water in clean vessels, so designed and placed that they cannot readily become polluted, and the feeding of a good ration in clean hoppers and troughs. Water troughs and mash hoppers should be equipped with reels and stands. Scratch grain should be fed in long V-shaped troughs; litter feeding of scratch is unsanitary. Grain troughs should be hung up when not in use so as to prevent unnecessary soilage.

The colony brooder house, like the laying house, should be of ample size for the number of birds for which it is intended, properly insulated if intended for early brooding, well ventilated but free from drafts, and dry. The house should have a double-boarded floor to ensure floor warmth and freedom from floor drafts, and should be built on a pair of good skids to facilitate moving. The house equipment should include: chick guard, mash and grain troughs of various sizes, waterers on wire stands, and forcing roosts that have wire underneath to prevent the chicks from coming into contact with their droppings. (Plans for the construction of a brooder house, a range shelter and poultry equipment may be obtained from the Department of Animal Science, University of Alberta, Edmonton.)

The brooder house should be given a thorough cleaning (similar to that outlined for the fall cleaning of a laying house) in the fall after the young stock has been transferred to winter quarters. Following cleaning, disinfection and whitewashing, the brooder house should be moved into position on the piece of ground that is to be used for range the next season.

The floor of the brooder house should be kept well covered with clean, dry litter. During the first two weeks of brooding, the litter (especially under the hover) should be removed daily. Later, weekly cleaning will suffice, and when the birds are staying outside most of the day, cleaning every two weeks will be found adequate.

As with layers, nothing is more important than the providing of clean, fresh water in clean vessels and the feeding of a good ration in clean hoppers and troughs. Mash troughs for young chicks should be equipped with reels and stands in order to prevent feed soilage. Range mash hoppers for older chicks should be weather-proof and should be moved a short distance at each filling so as to avoid excessive contamination of the ground in one place. During the first eight to ten weeks of brooding, scratch grain should be fed in troughs after which period it can be better supplied in range hoppers. Water vessels should be provided with low wire-covered stands so as to prevent the chicks from coming into contact with the damp, dropping-polluted, germ-infested litter or ground usually present around waterers.

The ground over which poultry range should be kept clean. This is necessary since many disease-producing germs and parasites to which poultry are subject are soil borne. The easiest way to ensure clean ground for poultry is to follow a system of range rotation. A two-year rotation is recommended for adult stock; a three-year rotation for young stock. In the former, the ground around the laying house is so fenced as to provide for two yards of approximately equal size. These yards are used in alternate years. The yard not occupied in any one year is cleaned by frequent cultivation or by cultivating for garden use. In the three-year rotation for young stock, three strips of range are required. Each piece of ground is used one year and rested two years. During the first year of rest the ground is cultivated frequently. Alfalfa or other range is sown in the spring of the second year of rest. If the range is not grazed too heavily, cultivation and re-seeding at less frequent intervals would prove satisfactory. The strips of ground do not have to be fenced but should be far enough apart to prevent the birds from running over the previous year's range.

Besides using a system of range rotation there are a number of other precautions that should be taken in order to ensure clean

ground for poultry. Firstly, adult chickens should not be permitted access to the young stock ranges. Adult stock are carriers of disease germs and would contaminate the young stock ranges. Secondly, chickens should not have access to turkey ranges and vice-versa. This is necessary in order to control blackhead and gapeworms (see Blackhead and Gapeworm). Thirdly, every effort should be made to keep the ranges clean and sanitary; low, wet spots should be filled or fenced so as to lessen chances of parasitic and coccidial infestation (see Internal Parasites and Coccidiosis); the ground around the brooder house should be swept each time the house is cleaned and the brooder house should be moved a short distance every four weeks to avoid excessive contamination of the ground in one place.

Under conditions where rotation of ranges is not possible, the use of wire floored sun-porches, or small fenced yards kept clean by changing the top three or four inches of soil yearly and cultivating frequently, is recommended. It is much better to confine poultry to a small piece of clean ground that is kept clean by frequent cultivation, than to let poultry range at large over dirty ground.

**Diagnosing Common Ailments.**

Most of the ailments to which poultry are susceptible can be readily recognized on case history, symptoms, and post-mortem findings.

Case history often proves of value in ascertaining the cause of trouble. A knowledge of such factors as the ration fed, feeding routine and whether or not the birds are confined or are on good range, frequently permits one to arrive at a more accurate diagnosis than one otherwise could make on symptoms and post-mortem findings alone. It often offers a means of confirming one's diagnosis and for this reason should not be overlooked.

When endeavouring to determine what is wrong with a bird, first take note of the symptoms displayed. Next, make a systematic examination of the bird for outward evidences of the presence of disease. Then make a similar examination of the bird for internal evidence of disease. Finally, diagnose the trouble in the light of the case history. Tables I and II will be found of assistance in arriving at a diagnosis. The reader is referred to the pages that follow for details on the various diseases.

(The Province of Alberta provides a free poultry disease diagnostic service. Farmers who wish to use this service should take or ship their ailing or dead specimens to the Poultry Pathologist, Provincial Veterinary Laboratory, Edmonton, Alberta. A letter similarly addressed, giving details on case history and symptoms, should accompany or follow the shipment.)

TABLE I

EXTERNAL AND INTERNAL SYMPTOMS IN YOUNG BIRDS

| DISEASE | SYMPTOMS | |
|---|---|---|
| | External | Internal |
| Fowl Pox | Wart-like lesions on comb, face, wattles, earlobes and eyelids. | Canker-like patches in mouth and throat. |
| Infectious Bronchitis | Watery eyes, sneezing, labored breathing, wheezing, coughing, choking, high mortality. | Purulent exudate in trachea (windpipe). |
| Newcastle Disease | Respiratory distress, paralysis of legs, tremor of head or body, incoordination of neck muscles, walking in circles or backwards. | Cloudiness and thickening of air sac membranes and membranes of abdominal cavity. |
| Lymphomatosis and Associated Conditions | Paralysis of legs, wings and neck, depigmentation of iris of eye, derangement of pupil of eye and blindness, irregular enlargement of legs, paleness about head. | Swollen and discolored sciatic (leg) and brachial (wing) plexuses (nerves), enlarged liver, spleen and kidneys, tumor-like masses in visceral organs, irregular enlargement of the bones, watery blood. |
| Pullorum | Droopiness, plaintive chirping, diarrhoea with pasting up behind, high mortality. | Nodules in heart and lungs, unabsorbed yolk. |
| Paratyphoid | Dullness, listlessness, weakness, sagging wings, diarrhoea. | Enlargement of liver and spleen, cheesy plugs in caeca, inflammation of intestine. |
| Erysipelas | Weakness, listlessness, loss of appetite, yellowish or greenish diarrhoea, bluish head, swollen snood in turkeys, heaviest losses amongst males. | Blotchy haemorrhages in superficial muscles, thick mucus in nasal passage, enlargement and congestion of liver, spleen and kidneys, catarrhal exudate in intestine. |
| Coryza | Watery eyes, sneezing, serous or thick foul-smelling discharge from nostrils, swollen sinuses, cheesy material in mouth cleft, labored breathing, wheezing. | None. |

| | | |
|---|---|---|
| Chronic Respiratory Disease | Decreased feed consumption, sneezing, hacking cough, breathing rattle, gasping for breath, watery eyes, nasal discharge, swollen sinuses, listlessness, loss of weight. | Frothy mucus in trachea (windpipe), bronchial tubes and air sacs; cloudy or yellowish air sacs, caseous material in air sacs, false membrane over liver, cheesy deposits in body cavity. |
| Infectious Synovitis | Paleness about head, shrivelled comb, leg weakness, unsteady stilted gait, swollen hocks, emaciation, greenish diarrhoea. | Tissue dehydration, enlarged spleen, caseous material in bursa, creamy or cheesy material in hock swellings, reduced hemoglobin content of blood. |
| Omphalitis | Pus about navel, puffy abdomen, diarrhoea. | Putrefaction about navel, unabsorbed yolk, distended gall bladder. |
| Botulism | Sleepiness, limpness of neck and wings, unsteady gait, prostration, looseness of feathers. | None. |
| Coccidiosis | Loss of appetite, emaciation, droopiness, passage of blood in droppings. Also as in Table II. | Enlarged caeca (blind pouches of intestine), thickening and inflammation of walls of caeca, blood or blood-stained material in caeca. Also as in Table 11. |
| Blackhead | Loss of appetite, poor growth, increased thirst, emaciation, droopiness, orange brown or green diarrhoea. | Ulcer-like areas of pink or yellowish color in liver, enlarged caeca, thickening and ulceration of walls of caeca. |
| Hexamitiasis | Listlessness, plaintive chirping, foamy watery diarrhoea, loss of weight. | Catarrhal inflammation of duodenum (U-shaped part of intestine), foamy mucus in intestine. |
| Trichomoniasis | Loss of appetite, haggard appearance, sagging wings, drooling from mouth, depressed chest, yellowish foamy diarrhoea. | Inflammation of gullet, crop and proventriculus (glandular stomach), grayish or yellowish pyramid-like necrotic areas in gullet and crop. |
| Aspergillosis | Droopiness, loss of appetite, emaciation, sneezing, wheezing, gasping for breath. | Grayish or greenish lesions in trachea, bronchi, lungs and air sacs. |
| Thrush | Loss of appetite, emaciation, labored breathing, ulceration of vent. | Cankerous growths in mouth, ulceration of gullet, crop, proventriculus, gizzard and intestine. |
| Nutritional Roup | Loss of appetite, poor growth, staggering gait, soreness of eyes, drowsiness, diarrhoea. | White deposits in kidneys and cloaca (chamber just inside vent) and on internal organs. |
| Riboflavin Deficiency | Paralysis of legs, inward curling of toes. | None. |

TABLE I—*Continued*

| DISEASE | SYMPTOMS | |
|---|---|---|
| | External | Internal |
| Rickets | Staggering gait, reluctance to stand or move, enlarged joints, bent legs, soft beaks, crooked breast bones, poor growth, general unthrift. | Beaded ribs, piling up of cartilage at ends of long bones. |
| Perosis | Bowing or twisting of legs, slipping of Achilles' tendon (large tendon at hock joint) from its normal position, enlargement and flattening of hock joint. | None. |
| Gout | Bluish comb, unsteady gait, lameness, swollen joints, diarrhoea. | Enlarged kidneys, white deposits on internal organs. |
| Haemorrhagic Disease | Haemorrhages around eyes and on the feet. Occasionally bloody droppings. | Haemorrhage in breast and thigh regions and in brain coverings and internal organs. |
| Beak Necrosis | Accumulation of mash around mouth, deformed beak. | None. |
| Impaction of the Crop | Loss of appetite, general distress, crop distended and doughy to feel. | Fibrous material in crop. |
| Pendulous Crop | Enlarged baggy crop. | None. |
| Impaction of the Gizzard | Loss of appetite. | Fibrous material in gizzard. |
| Bumblefoot | Swelling of ball of foot. | None. |
| Cannibalism | Toes or tail bleeding, feathers missing on back and abdomen, vent bleeding, disembowelling. | None. |
| Lice | Straw colored elongated insects found on bird at any time; bodies show head, thorax and abdominal divisions. | None. |

| | | |
|---|---|---|
| Common Chicken Mite | Gray or red ovoid insects found on bird at night. | None. |
| Northern Fowl Mite | Gray or red ovoid insects found on surface of feathers of bird in localized areas at any time. | None. |
| Depluming Mite | Feathers absent in certain areas. | None. |
| Large Intestinal Roundworm | Paleness of head and shanks, loss of appetite, poor growth, emaciation, droopiness, paralysis. | Long, white, unsegmented worms in intestine. |
| Caecal Worm | As in Large Intestinal Roundworm. | Small, white unsegmented worms in caeca. |
| Gapeworm | Inactivity, gasping for breath, death by suffocation. | Reddish-brown Y-shaped worms in trachea (windpipe). |
| Tapeworm | As in Large Intestinal Roundworm. | Long or short segmented worms in intestine. |

TABLE II

EXTERNAL AND INTERNAL SYMPTOMS IN ADULT BIRDS

| DISEASE | SYMPTOMS | |
|---|---|---|
| | External | Internal |
| Fowl Pox | As in Table I. | As in Table I. |
| Infectious Laryngo-tracheitis | Watery eyes, sneezing, labored breathing, wheezing, coughing, choking, high mortality. | Blood-stained exudate in trachea (windpipe). |
| Infectious Bronchitis | As in Table I. | Catarrhal exudate in trachea. |
| Newcastle Disease | Sudden onset, depression, droopiness, coughing, gasping, paralysis of legs, wings or neck, rapid decline in egg production, soft shelled eggs. | Cloudiness of air sac membranes, hardened egg yolk in body cavity. |
| Lymphomatosis and Associated Conditions | As in Table I. | As in Table I. |
| Monocytosis | Sudden onset, depression, loss of appetite, whitish watery diarrhoea, bluish comb, shrivelled legs, severe decline in egg production. | Spotty liver, chalk-white pancreas, haemorrhages on internal membranes, tenacious mucus in intestines, urate deposits in kidneys, fish-like flesh on breast. |
| Fowl Tuberculosis | Extreme loss of weight, lameness, swollen joints, greenish diarrhoea, paleness about head. | Grayish or yellowish raised tubercules of cheesy consistency in liver and spleen and on intestines. |
| Pullorum | None. | Misshapen, greenish or leaden colored yolks in ovary, enlarged and noduled heart, pericarditis (inflammation of the heart sac). |
| Fowl Typhoid | Droopiness, sleepiness, paleness about head, greenish or yellowish diarrhoea, high fever, increased thirst. | Greenish bronze liver, enlarged spleen and kidneys, misshapen greenish or leaden colored yolks in ovary, enlarged and noduled heart, pericarditis. |

| | | |
|---|---|---|
| Paratyphoid | Loss of appetite, unthrift, emaciation, unkempt appearance, diarrhoea. | As in Table I. |
| Fowl Cholera | Sudden death, loss of appetite, droopiness, sleepiness, darkness about head, greenish or yellowish diarrhoea, high fever, increased thirst, swelling of wattles. | Sticky mucus in mouth, congestion of visceral organs, friable liver, pinhead sized necrotic areas in liver, small haemorrhages on internal organs. |
| Erysipelas | As in Table I. | As in Table I. |
| Coryza | As in Table I. | As in Table I. |
| Chronic Respiratory Disease | As in Table I. Gradual and prolonged drop in egg production. | As in Table I. |
| Botulism | As in Table I. | As in Table I. |
| Coccidiosis | Loss of appetite, progressive emaciation, sluggishness, paleness about head, paralysis of legs. Also as in Table I. | Inflammation and thickening of walls of the intestine. Also as in Table I. |
| Blackhead | Progressive emaciation, sunken eyes, sluggishness diarrhoea. | As in Table I. |
| Hexamitiasis | As in Table I. "Carriers" look normal. | As in Table 1. "Carriers" look normal. |
| Trichomoniasis | As in Table I. | As in Table I. |
| Aspergillosis | As in Table I. | As in Table I. |
| Favus | Dirty white or yellowish scaly lesions on comb, earlobes, face and wattles. | None. |
| Thrush | As in Table I. | As in Table I. |
| Nutritional Roup | Low hatchability, decreased egg production, loss of appetite, incoordinated gait, soreness of eyes, swollen sinuses, droopiness. | White deposits in kidneys and cloaca and on internal organs, white pustules in mouth, pharynx and gullet. |
| Riboflavin Deficiency | Low hatchability, decreased egg production. | None. |

TABLE II—*Continued*

| DISEASE | SYMPTOMS | |
|---|---|---|
| | External | Internal |
| Rickets | Low hatchability, decreased egg production, production of thin shelled abnormally small eggs; weak and crooked bones. | None. |
| Gout | As in Table I. | As in Table I. |
| Obesity | Sluggishness, excess weight. | Excess of abdominal fat. |
| Beak Canker | Sore at beak angle. | None. |
| Impaction of the Crop | As in Table I. | As in Table I. |
| Pendulous Crop | As in Table I. | As in Table I. |
| Impaction of the Gizzard | As in Table I. | As in Table I. |
| Bumblefoot | As in Table I. | As in Table I. |
| Ascites | Distended abdomen. | Serous fluid in body cavity. |
| Ruptured Yolk | Dark comb, sagging of abdomen. | Yolk material in body cavity. |
| Intraovulation | Dark comb, sagging of abdomen. | Yolks in body cavity. |
| Impacted Oviduct | None. | Hardened yolk and albumen in oviduct (egg tube). |
| Ruptured Oviduct | None. | Break in wall of oviduct. |
| Internal Laying | Sagging of abdomen. | Eggs and egg matrial in body cavity. |

| | | |
|---|---|---|
| Egg Bound | Straining to lay. | Egg in lower part of oviduct. |
| Prolapsus | Eversion of cloaca (chamber just inside vent) and lower oviduct. | None. |
| Cloacitis | Swelling, inflammation and ulceration of vent, obnoxious odour. | Inflammation and ulceration of cloaca. |
| Cannibalism | As in Table I. | As in Table I. |
| Lice | As in Table I. | As in Table I. |
| Common Chicken Mite | As in Table I. | As in Table I. |
| Northern Fowl Mite | As in Table I. | As in Table I. |
| Scaly Leg Mite | Raised scales on legs. | Ovoid insects under scales. |
| Depluming Mite | As in Table I. | As in Table I. |
| Large Intestinal Roundworm | Decreased egg production, loss of weight, unthrift. | As in Table I. |
| Caecal Worm | As in Large Intestinal Roundworm. | As in Table I. |
| Gapeworm | As in Table I. | As in Table I. |
| Tapeworm | As in Large Intestinal Roundworm. | As in Table I. |

## VIRUS DISEASES

A number of the more troublesome diseases to which poultry are susceptible are caused by filterable viruses. (Filterable viruses are disease-producing agents which are so small as to be invisible under the ordinary microscope). Fowl pox, infectious laryngotracheitis, infectious bronchitis, Newcastle disease, lymphomatosis and avain monocytosis are among the more important diseases in this group.

### Fowl Pox.

Fowl pox, also known as chicken pox or avain diphtheria, is a highly infectious disease, to which both chickens and turkeys are susceptible. This disease is encountered most frequently during the fall and winter months, and is more prevalent at the Pacific coast than it is in the Prairie and Eastern provinces of Canada.

Fowl pox may manifest itself in one or both of two forms—the skin form and the diphtheritic form. The skin form is characterized by brownish wart-like lesions on the comb, face, wattles, earlobes and eyelids (see Fig. 1). The diphtheritic form is characterized by

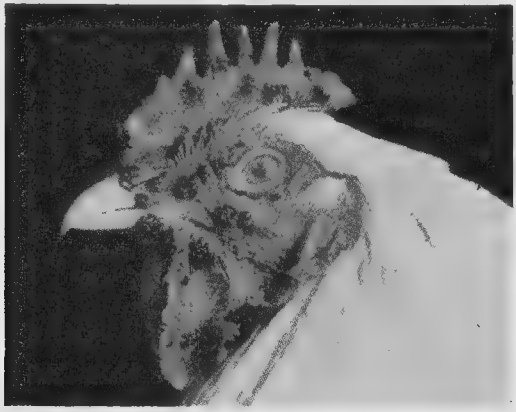


**Fig. 1—Wart-like lesions on comb, face, wattles and earlobe of a fowl affected with fowl pox.** (After Barger and Card.)

canker-like patches in the mouth and throat. Both forms may be accompanied by loss of appetite, loss of weight and decreased egg production. Post-mortem examination reveals no internal changes. Diagnosis is usually made on the basis of pox lesions and the infectious nature of the malady, although inoculation experiments offer the only means of proving the presence of this disease.

Feed and water constitute the principal agents responsible for the spread of fowl pox in a flock. The virus causing the disease gets into the feed and water while affected birds feed and drink.

Later, it gains entrance to the bodies of healthy birds through skin and mouth abrasions.

Mortality from this disease varies greatly, depending on the severity of the outbreak. In most outbreaks, mortality losses are light, but financial losses through delayed egg production in pullets and decreased egg production in laying hens are great.

Where an outbreak of fowl pox has occurred, the following control measures are recommended:

(1) Remove and isolate all affected birds.
(2) Thoroughly clean and disinfect the houses and equipment.
(3) Vaccinate apparently healthy birds. If it is a flock of vigorous growing birds that is affected, use chicken pox vaccine for vaccination. It induces solid immunity. If it is a flock of none-too-vigorous growing birds or a flock of laying birds, use pigeon pox vaccine. It is less severe than chicken pox vaccine. Pigeon pox vaccine induces only temporary immunity.
(4) Remove and isolate any other birds showing evidence of infection.

Many of the birds removed from the flock may be saved by proper treatment. Treatment entails removing the wart-like lesions and cankerous growths and painting the raw areas with 5% iodine, 10-15% argyrol or 4% silver nitrate solution. Before removal, the wart-like lesions should be softened with warm water to which a small amount of salt has been added. Birds that survive an attack of fowl pox are in most cases immune to further infection from this disease.

Once fowl pox has appeared on a farm, yearly vaccination of young stock with chicken pox vaccine should be practised. The best time to vaccinate growing chickens and growing turkeys is when they are eight to sixteen and ten to twelve weeks old respectively. Under no circumstances should unthrifty stock be vaccinated. If such is done, in all probability an outbreak of the disease will occur.

Two methods are commonly employed in connection with vaccination for fowl pox, namely, the follicle and stick methods. The follicle method is used with pigeon pox vaccine on laying and none-too-vigorous growing birds. It consists of removing six or eight feathers in the upper thigh region and brushing into the follicles a small amount of a water suspension of the virus. The stick method, on the other hand, is used with chicken pox vaccine on vigorous growing chickens and on all classes of turkeys. The method consists of piercing the wing web with the eye ends of two fine needles which have been previously dipped in vaccine. To

carry out the stick method, a three by one-half inch test tube provided with a rubber stopper into which are inserted, with eye ends out, two fine needles, is required. With both methods of vaccination "takes" (presence of a scab) are read 7 - 10 days after vaccination (see Fig. 2). Birds that do not show "takes" should be revaccinated. Immunity develops in four weeks.

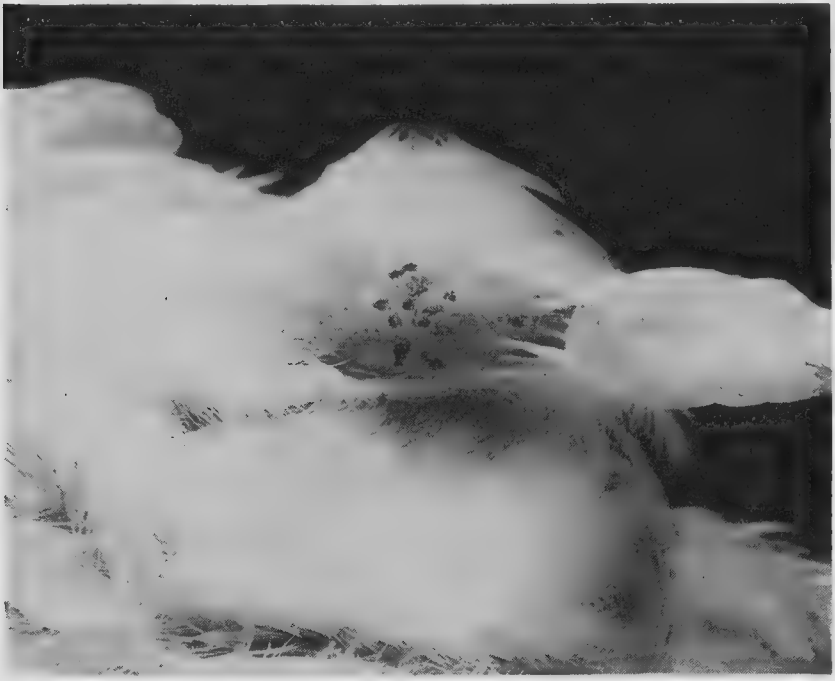


**Fig. 2—A "take" following successful vaccination for fowl pox by the follicle method is evidenced by the development of brownish scabs in from seven to ten days.** (After Beach.)

Flocks which have not been exposed to fowl pox should not be vaccinated unless fowl pox is prevalent in the district.

**Infectious Laryngotracheitis.**

This is an acute, very infectious disease to which chickens are highly susceptible. Generally speaking, the disease is more common in adult chickens than in young chickens. Outbreaks of laryngotracheitis occur mainly during the fall and winter months. This disease is more prevalent in the Prairie and Eastern provinces of Canada than it is at the Pacific coast.

The first symptoms of this disease are watery eyes and sneezing. As the disease progresses, breathing becomes more difficult and wheezing and rattling sounds are audible. During coughing spells

**Fig. 3—Typical posture of fowl during inspiration of air in advanced stages of infectious laryngotracheitis.** (After Stover.)

that follow, afflicted birds shake their heads in an effort to dislodge mucous exudate that accumulates in the larynx and trachea (windpipe). In advanced stages, affected birds take on a sitting posture extending their heads during each inspiration of air (see Fig. 3). Mortality is usually highest on the third or fourth day after exposure to the disease. The disease runs its course in from 7-15 days, although symptoms may be manifested for longer periods than this. Birds that recover from the disease harbour the causative virus in their throats for months, and may thus serve as a source of infection to healthy birds.

Post-mortem examination shows mucus about the nostrils and the presence of bloody exudate in the larynx and trachea. Small haemorrhages are also seen in the larynx and trachea. In some instances, a yellowish cheesy exudate may be found in the larynx and trachea. Diagnosis of laryngotracheitis is usually made on the aforementioned symptoms and post-mortem findings and on the rapidity of spread of the infection. Positive proof of the presence of laryngotracheitis infection, however, can only be obtained by carrying out inoculation experiments.

Laryngotracheitis may be introduced into a flock through the purchase of "carrier" birds, or by bringing contaminated shipping crates, feed sacks, etc., on the premises. The disease is spread from one bird to another through the air.

Laryngotracheitis is considered to be one of the most serious diseases with which poultrymen have to contend. Very heavy losses, both in birds and egg production are often sustained when this malady appears.

Success in getting an outbreak of laryngotracheitis under control depends to a considerable extent on the stage which the disease has reached when detected. At the first sign of this trouble, all sick birds should be removed from the flock and the houses and equipment should be thoroughly cleaned and disinfected. The virus is readily destroyed by ordinary disinfectants. Further cases should be watched for and removed from the flock as soon as noticed. Birds removed from the flock should be disposed of immediately by burning. It is useless to treat affected birds, for even if treatment should prove successful, the recovered birds cannot be re-admitted to the flock for they retain the causative virus in their throats long after recovery. In flocks where the disease has a good start, all birds that have been exposed to the disease should be disposed of, and the houses and equipment should be thoroughly cleaned and disinfected. No poultry should be housed in the depopulated buildings until at least three months time has elapsed.

Since laryngotracheitis is introduced into a flock by mechanical means, i.e., on the shoes or clothes of visitors, on returned shipping crates, on feed sacks, etc., or through the importation of diseased or "carrier" stock, measures of prevention obviously must entail consideration of these factors. Visitors who have recently had direct contact with this disease should not be allowed on the premises. The handling of birds by visitors should be discouraged. Shipping crates should be washed and disinfected immediately on arrival back on the premises. Feed sacks should be thoroughly disinfected before being used for hauling feed about the farm. New stock should only be purchased from flocks that have never experienced this disease.

In recent years, vaccination against laryngotracheitis has been introduced. In vaccinating for laryngotracheitis, the vaccine is applied to the mucous membranes of the cloaca and bursa through the vent by means of a swab (see Fig. 4). "Takes" (presence of inflammation and a false membrane in the cloaca and bursa) are read four or five days after vaccination. Birds that do not show "takes" should be revaccinated. Immunity develops in 4 - 9 days. Best results are obtained when birds are vaccinated at 2 - 4 months of age.

**Fig. 4—Showing the cloacal method of vaccinating fowls for the prevention of infectious laryngotracheitis.**

It should be pointed out that in flocks where laryngotracheitis has never occurred and in flocks where the disease has occurred and where it has been brought under control by depopulation, vaccination should not be practised. Vaccination should only be resorted to on farms where infection is known to exist, that is, on farms where an outbreak has occurred and where depopulation was not adopted in bringing it under control.

### Infectious Bronchitis.

This is a highly infectious respiratory disease of chickens. The disease is caused by a virus that is immunologically distinct from the virus that causes infectious laryngotracheitis.

The disorder is of widespread occurrence, frequently being noted in hatcheries and broiler plants.

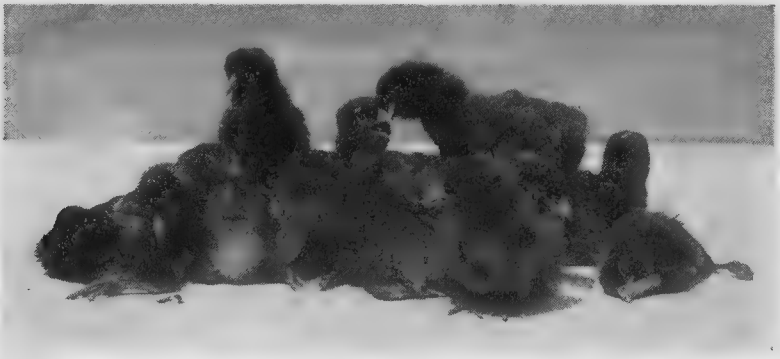


**Fig. 5—Infectious bronchitis in 4-day-old chicks.** (After Illinois Agricultural Experiment Station.)

Chicks between the ages of two days and three weeks are highly susceptible to this disease, although older birds may also be affected. The symptoms (see Fig. 5) in infectious bronchitis are similar to those referred to under Infectious Laryngotracheitis. Post-mortem examination of semi-mature and adult birds fails to show haemorrhages in the larynx and trachea like those seen in infectious laryngotracheitis. Variable amounts of catarrhal exudate are found in the larynx and trachea. In young chicks, small pin-point haemorrhages may be noted in the larynx and trachea, and there is usually a purulent exudate present.

While symptoms displayed, post-mortem findings and rapidity of spread of this disease are highly diagnostic, positive diagnosis of infectious bronchitis is dependent upon inoculation experiments.

The disease does not cause significant mortality in semi-mature and adult stock, but in young chicks, mortality from infectious bronchitis can be quite high. Financial loss resulting from depressed egg production in layers affected with infectious bronchitis is often quite serious.

Infectious bronchitis is disseminated in a manner not unlike that referred to under Infectious Laryngotracheitis. There is no specific treatment for infectious bronchitis. Control in areas such as Alberta, where the incidence of the disease is known to be high, is best effected through vaccination.

Only healthy birds should be vaccinated. Chickens of one day to four months of age may be vaccinated. It is not advisable to vaccinate laying birds as egg production is apt to be affected adversely for a prolonged period of time. Immunity to infectious bronchitis usually lasts for one year; accordingly, it is not necessary to revaccinate at frequent intervals to ensure immunity as is the case with Newcastle disease. As an aid to getting birds through the vaccination period without serious losses from secondary infections it is recommended that a high level (see Chronic Respiratory Disease) of antibiotic be included in the feed for a period of about a week following vaccination.

Several methods of applying infectious bronchitis vaccine are available. The vaccine may be applied as liquid spray, dust spray, drinking water additive or as an intranasal-intraorbital vaccine. The first three methods of application are perhaps the most widely used. For specific details on the method of applying the various types of vaccine, the reader is referred to the manufacturers instructions.

Attention should be drawn here to the desirability of adopting a bronchitis vaccination program in flocks that are to be used for breeding purposes. Chicks from such vaccinated flocks possess a

temporary parental immunity to infectious bronchitis and because of this are less apt to be affected adversely by vaccination with infectious bronchitis vaccine during the first week or two after hatching. In addition chicks that possess parental immunity to such diseases as infectious bronchitis and Newcastle disease appear to be less prone to developed chronic respiratory diseases than chicks not possessing such temporary parental immunity (see Chronic Respiratory Disease).

**Newcastle Disease.**

Newcastle disease is a highly infectious virus disease to which chicks and adult chickens are very susceptible. In England and Europe the disease is characterized by acuteness and high death rate. In America, on the other hand, the malady is usually milder in character and less fatal.

Outbreaks of Newcastle disease have been reported from most areas of the United States and Canada. In so far as Canada is concerned, relatively few outbreaks have been reported in the Prairie Provinces while numerous outbreaks have occurred in British Columbia and in Ontario.

Chicks affected with the American form of Newcastle disease exhibit respiratory distress similar to that noted in infectious bronchitis. In addition nervous affliction as evidenced by partial or complete paralysis of one or both legs (see Fig. 6), tremor of the head or whole body, and incoordination of action of the neck muscles are frequently noted (see Fig. 6). Affected chicks may walk in circles or backwards.

In laying birds the onset of the disease is sudden. Depression and droopiness spread rapidly through the flock. Within a week

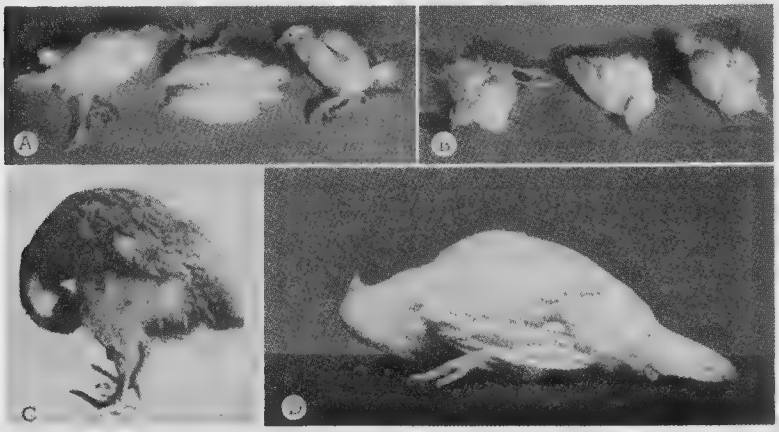



**Fig. 6—Newcastle disease. (A)—paralysis of legs and wings; (B)—paralysis of legs and incoordination of neck muscles; (C)—incoordination of neck muscles.** (After Hoffman); **(D)—incoordination of neck muscles.** (After Stover.)

most of the birds in the flock may be sitting around on the roosts or floor. Some of the birds may be coughing and gasping in a manner not unlike that noted in infectious laryngotracheitis and infectious bronchitis. A few of the birds may show nervous affliction as evidenced by paralysis of the legs, wings or neck. Feed consumption decreases rapidly. Egg production generally declines abruptly until the birds are completely out of production. Many soft shelled eggs are laid off the roost and on the floor. Recovery is usually rapid. After three or four weeks the birds start laying again. However, for a considerable period, many odd shaped, poor shelled, low quality eggs are produced.

Internal examination of chicks shows cloudiness and thickening of the air sac membranes and membranes of the abdominal cavity. Similar examination of adult chickens frequently reveals no lesions. In some instances the air sac membranes may be clouded and liquid or hardened egg yolk may be found in the abdominal cavity.

Presumptive diagnosis may be based on external and internal symptoms, however, positive diagnosis should only be made on the basis of findings, in the laboratory. Farmers suspecting the presence of this disease in their flocks are advised to take or send specimens to the Provincial Veterinary Laboratory, Edmonton, for examination.

Newcastle disease may be spread from one farm to another by traffic in sick or "carrier" birds. Spread by wild birds and contaminated chicken crates or feed sacks is also indicated.

Mortality from the American form of Newcastle disease is generally light being somewhat greater in chicks than in adult birds. Losses through depressed growth rate in broiler flocks and through cessation of egg production in laying birds, however, may be serious.

There is no treatment for Newcastle disease. In Alberta, as previously indicated, the incidence of the disease is slight. In addition poultry flocks are widely separated. Under such conditions it would appear practical, in the event of an outbreak of Newcastle disease, to attempt to prevent further losses from this disease by depopulating the premises of all birds and cleaning and disinfecting the house and equipment rather than resorting to vaccination which would seem to be the most satisfactory mode of controlling this disease in areas where the disease is of common occurrence and where poultry flocks are more concentrated.

In the event that control of losses from Newcastle disease through vaccination appears advisable, certain precautions and procedures should be observed. Only healthy birds should be vaccinated. Vaccination of birds of low vigor is likely to result in high mortality. Young stock should be protected against Newcastle disease by vaccination at one to ten days of age. Young stock to be used for

laying purposes should be revaccinated at about four to five months of age and vaccination again at nine to eleven months of age is advisable if they are to be used as breeders. Immunity to Newcastle disease, as a result of vaccination, only lasts three to five months. It is for this reason that it is necessary to revaccinate periodically to control losses from the disease.

Newcastle disease vaccine may be applied as a liquid-spray vaccine, dust-spray vaccine, drinking-water type vaccine, intranasal-intraorbital vaccine or as a wing-web vaccine. The first three methods of application are perhaps the most widely used. For specific details on the use of the different types of Newcastle vaccine the reader is referred to the manufacturers directions.

**Lymphomatosis and Associated Conditions.**

Until recently the term "fowl leukosis" has been used to designate a widespread disease-complex which, depending on the symptoms displayed, has been referred to in common terms as fowl or range paralysis, pearl eye or iritis, big liver disease, big bone disease and leukemia. It now appears that several different viral agents are involved and that, for the present at least, it would be advisable to deal with the complex under the heading lymphomatosis and associated conditions, the former to include neural, ocular and visceral lymphomatosis; the latter, osteopetrosis and fowl leukemia. Evidence obtained to date would seem to indicate that visceral lymphomatosis is caused by a different virus than the viral agent or agents responsible for neural and ocular lymphomatosis. It also appears to be different from the viruses that cause osteopetrosis and fowl leukemia.

*Lymphomatosis.*—As previously indicated the three forms of lymphomatosis are termed neural lymphomatosis (fowl or range paralysis), ocular lymphomatosis (pearl eye or iritis) and visceral lymphomatosis (big liver disease). The disease occurs primarily in chickens although it has been reported in turkeys.

Neural lymphomatosis affects the nerves causing paralysis of legs (see Fig. 7) wings or neck. Affected birds are often unable to walk properly and lie on their sides with one leg extended forward and the other backward. In other instances the wings may be affected causing them to droop (see Fig. 8). Internal examination of birds affected with this form of the disease reveals a swelling and yellowish discoloration of the nerves leading from the spinal column to one or both legs (see Fig. 9 and 10), to one or both wings (see Fig. 11) or to the neck. Occasionally tumor-like growths may be found in the ovary (see Fig. 11).

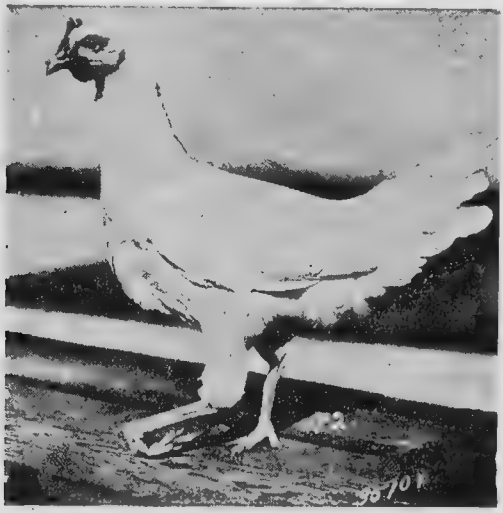


Fig. 7—Neural lymphomatosis. Paralysis of leg. (After Biely.)

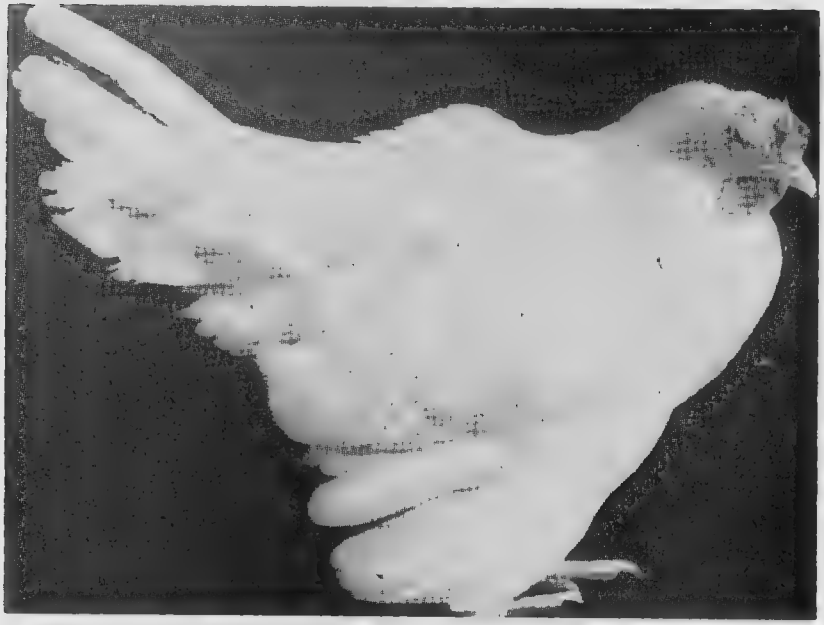


Fig. 8—Neural lymphomatosis. Note droopy wing. (After U.S.D.A.)

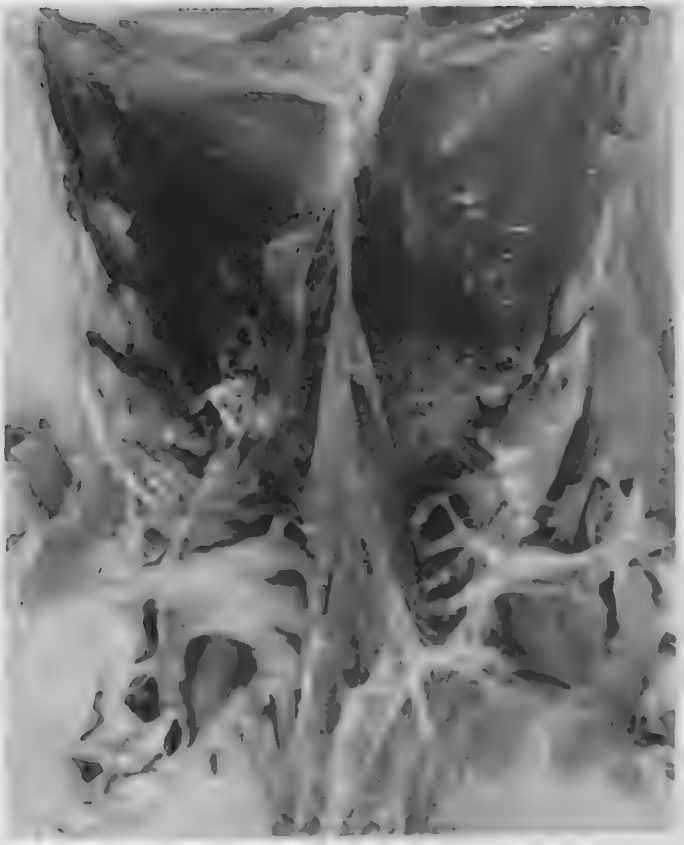


Fig. 9—Neural lymphomatosis. Note swollen left sciatic plexus as compared to normal nerve ganglion on the right. (After Beach.)

Fig. 10—Normal (A) and abnormal (B) sciatic nerves in fowl affected with neural lymphomatosis. (After California Agricultural Experiment Station.)

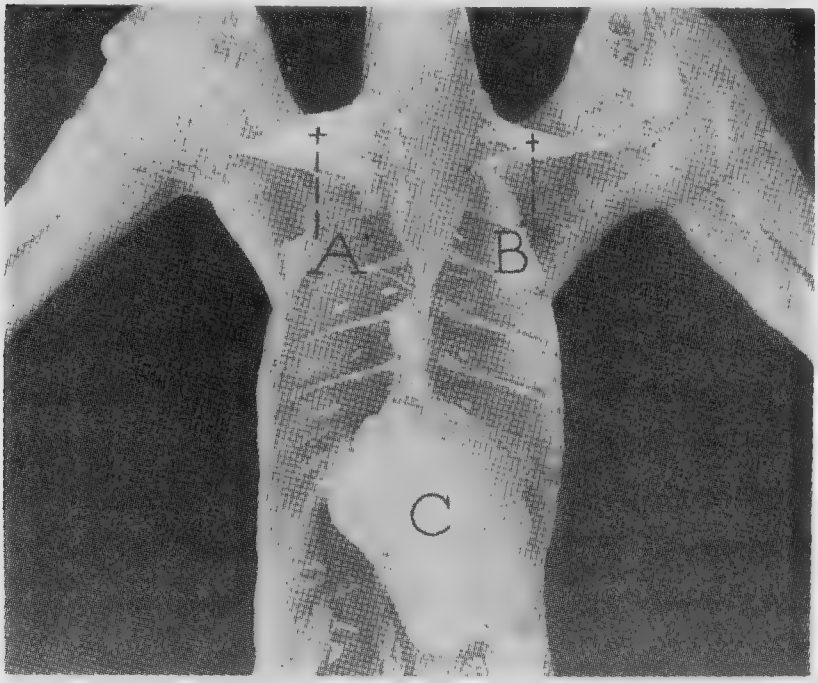



**Fig. 11—Neural lymphomatosis. Enlarged brachial plexus nerves (A and B) leading to the wings. The enlargement is more pronounced in the right wing (A) than in the left wing. Note also ovarian tumor (C).** (After U.S.D.A.)

In ocular lymphomatosis the iris of the eye frequently undergoes a change in color from the normal orange color to gray, hence the name "pearl eye". This gray iris color should not be mistaken for the pearl or gray eye color which is characteristic of certain breeds. In addition to the change in iris color the pupils of the eyes of affected birds are often distorted in shape and reduced in size (see Fig. 12). Affected birds frequently are blind.

There are usually no external symptoms in visceral lymphomatosis. On internal examination, however, the liver is often found to be greatly enlarged (see Fig. 13), hence the name "big liver disease". In addition the spleen and kidneys may be enlarged (see Fig. 14) and tumor-like masses may be found in the various visceral organs (see Fig. 15).

**Fig. 12—Ocular lymphomatosis. Upper left shows normal eye. Lower right shows pin-point pupil characteristic of the disease.** (After Iowa State College.)

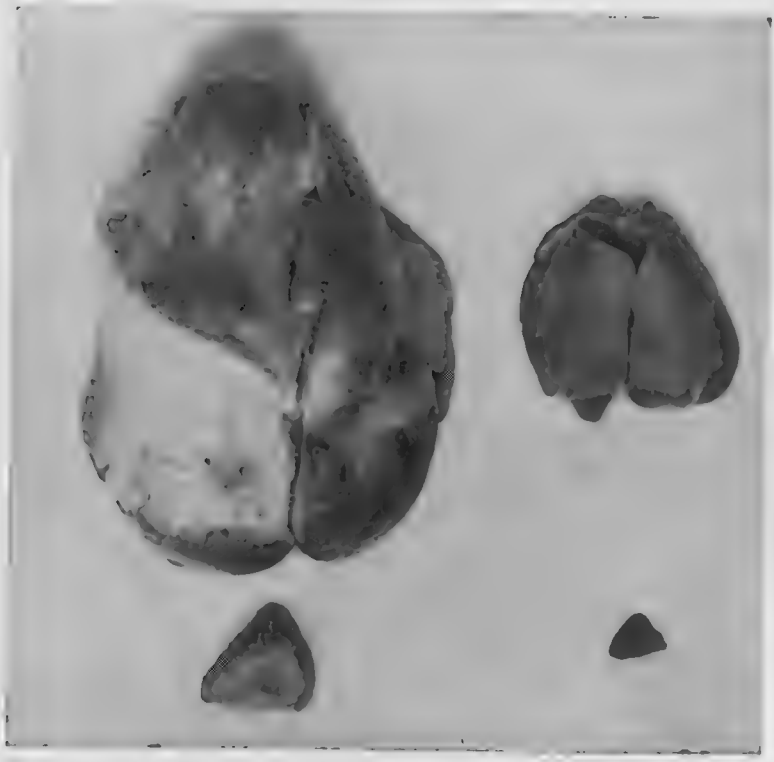


Fig. 13—Liver and spleen from a fowl affected with visceral lymphomatosis on the left. Normal liver and spleen on the right. (After Clandinin and Robblee.)

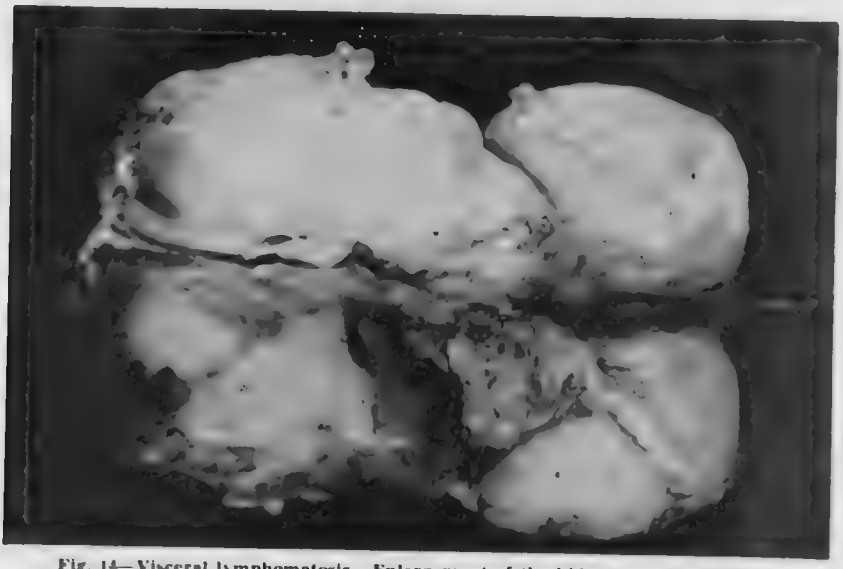


Fig. 14—Visceral lymphomatosis. Enlargement of the kidneys. (After Biely.)

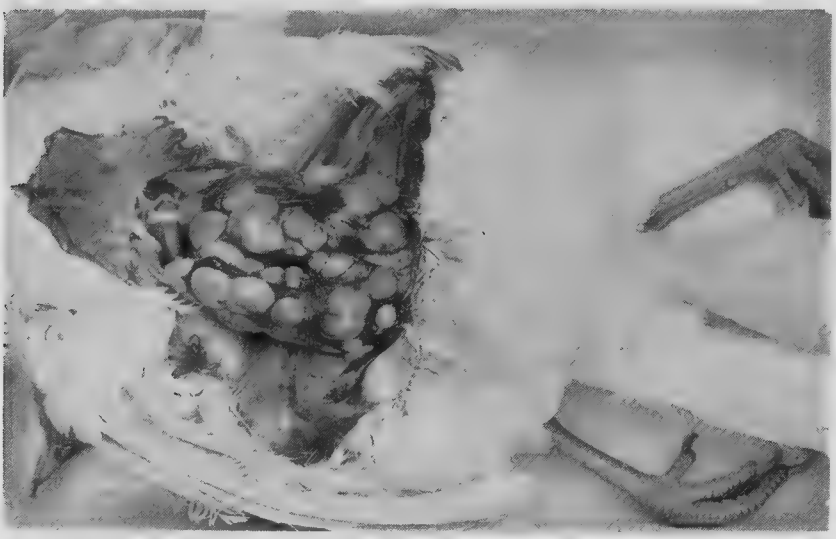


**Fig. 15—Visceral lymphomatosis. Note tumor-like masses in the liver.** (After Moynihan and Biely.)

*Associated diseases.*—As indicated earlier osteopetrosis and fowl leukemia are usually considered in discussions on this disease-complex. In osteopetrosis the bones of the body, particularly the leg and wing bones, are irregularly enlarged (see Fig. 16 and 17). In leukemia the principal changes are confined to the blood. In this regard, there is usually an increase in the number of immature red or white blood cells. Birds affected with this condition often develop a light yellow color about the head and an unthrifty appearance.

**Fig. 16—Osteopetrosis. Note thickening of legs.** (After Jungherr and Landauer.)

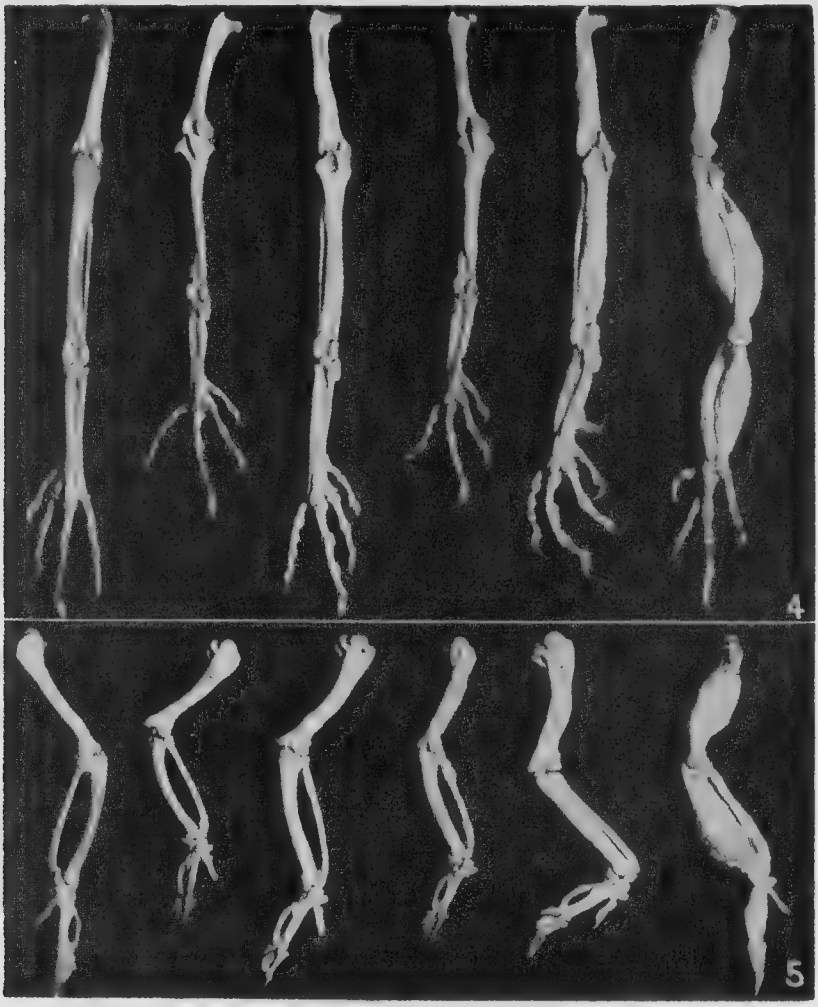



**Fig. 17—Irregular enlargement of long bones taken from fowls affected with osteopetrosis.** (After Jungherr and Landauer.)

Lymphomatosis is perhaps the most destructive disease with which poultrymen have to contend. It is estimated that this disease-complex results in an annual loss to poultrymen of America in excess of eighty million dollars. Approximately 75% of this loss can be attributed to visceral lymphomatosis.

The mode of transmission of lymphomatosis varies somewhat with the form of the disease. The neural form has not been shown to be transmitted from parent to chick through the egg but evidence has been obtained which indicates that this form of lymphomatosis

is spread by contact particularly during the early brooding period. The nearer chicks are brooded to adult chickens the higher is likely to be the incidence of this form of the disease in the growing stock. The mode of transmission of ocular lymphomatosis is not understood. Insofar as visceral lymphomatosis is concerned, it has been well established that this form of lymphomatosis is spread by contact and through hatching eggs. The virus is present in secretions of the oral and nasal passages and in the droppings of affected birds. It gains entrance to the bodies of healthy birds through exposed mucous membranes. With regard to egg transmission several important facts have been discovered. In the first place, many of the hens that pass the virus in their eggs do not show outward evidence of infection. Secondly, the progeny of "carrier" hens do not develop a high incidence of lymphomatosis owing to the fact that a certain amount of parental immunity has been conferred on them. Finally, it has been shown that egg transmission of the disease from chicks hatched from infected eggs to chicks from eggs of hens that have had no experience with the virus is of more importance than transmission of the disease from infected hens to their own chicks.

No treatment for lymphomatosis is known. The future holds promise of a vaccine, but until such is available for commercial application, losses from this disease-complex may be minimized by:

1. Breeding for resistance to lymphomatosis. On pedigree poultry farms, rearing the young stock in close proximity to the adult stock so as to ensure exposure to the disease and selecting for resistance to the disease on a family basis is recommended.

2. Obtaining hatching eggs, chicks and adult stock, if possible, only from strains of birds that have experienced heavy losses from lymphomatosis in previous years and in which the disease has been brought under control by the selection technique referred to above. Stock from such strains usually show a lower incidence of lymphomatosis than stock from strains that have not experienced the disease, since the latter have not had a chance to build up resistance to the disease.

3. Brooding and rearing young stock away from adult birds except, of course, on pedigree poultry farms where a selection program for disease resistance is in effect. In this regard, precaution should be exercised to avoid contact between chicks being raised for commercial egg production and adult stock since lymphomatosis is readily spread from adult "carrier" stock to chicks.

4. Disposing of all birds that show outward symptoms of the disease as soon as noticed. By following this procedure, spread of the malady can be partially checked.

**Avian Monocytosis.**

Avian monocytosis, also known as pullet disease, blue comb or fowl nephritis is a disorder that affects birds around the time that they are in full lay, although it has been reported to affect younger birds as well.

The exact cause of this disease is not known. It has been suggested that a metabolic disturbance resulting in urinary stagnation and subsequent bacterial infection of the kidneys contributes to the development of the abnormal kidney structure which characterizes the disorder. The specific cause of the metabolic upset is not, as yet, clear although a virus has been suggested and the housing of birds in buildings that are too warm and the feeding of new wheat have been singled out as possible contributing factors to outbreaks of this disorder.

The onset of avian monocytosis is sudden. Initial symptoms include depression, loss of appetite and the passage of whitish watery droppings. Affected birds are dark about the comb (hence name "blue comb") and their legs appear shrivelled and dehydrated. In laying flocks that are affected there is a severe drop in egg production. Internal examinations of affected birds reveals spotty livers, chalk-white pancreas, haemorrhages on the internal membranes, tenacious mucus in the intestine, urate deposits in the kidneys and fish-like flesh on the breast.

Presumptive diagnosis may be made on the aforementioned external symptoms and internal findings but positive diagnosis should only be made after complete laboratory examination. The latter usually involves histological examination of the kidneys and a study of the monocyte content of the blood.

Mortality from this disease may run as high as 20%. Economic loss through cessation of egg production may also be serious.

Birds affected with avian monocytosis should, if possible, be kept in cool, well ventilated quarters. Recently certain antibiotics have been shown to have beneficial value in connection with the treatment of birds affected with this disease. In this regard, the inclusion of 250 to 500 ppm of streptomycin sulphate, dihydrostreptomycin, penicillin G, diamine penicillin, procaine penicillin or dibenzlethylene-diamine penicillin G in the drinking water has given encouraging results. Terramycin at the rate of 1.5 grams of terramycin to each 10 gallons of drinking water has also proved highly effective in treating this disorder. Additions to the feed of bacitracin at the rate of 200 grams per ton of ration or penicillin, aureomycin, terramycin and streptomycin at the rate of 500 grams per ton of ration has been reported effective in reducing mortality from this disease.

## BACTERIAL DISEASES

Some of the most common and economically important diseases of poultry are caused by bacteria. (Bacteria are one-celled microscopic plants.) The more important diseases in this group are fowl tuberculosis, pullorum, fowl typhoid, paratyphoid, fowl cholera, erysipelas, coryza, chronic respiratory disease, infectious synovitis, omphalitis and botulism.

**Fowl Tuberculosis.**

Fowl tuberculosis is a chronic infectious disease that may be contracted at any age, but, because of its chronic nature, symptoms are usually not observed in birds less than one year old. The disease is more prevalent in the Prairie provinces than in the other provinces of Canada.

Tuberculosis in poultry is caused by the micro-organism *Mycobacterium tuberculosis avium.* Besides chickens and turkeys, other classes of livestock, such as swine, are susceptible to avian tuberculosis.

As previously stated, symptoms of tuberculosis in poultry are usually only apparent in birds that are over one year old. The principal symptoms of tuberculosis in poultry are: extreme loss of weight, lameness, swollen joints, diarrhoea, sunken face, paleness about the head, ruffled feathers and drowsiness.

Post-mortem examination of birds affected with tuberculosis reveals grayish or yellow tubercles in the liver, spleen and intestines (see Fig. 18). Occasionally similar nodules are found in the bones, trachea (windpipe), lungs, ovary, and mesenteries. The nodules themselves are of cheesy consistency and in the liver and spleen may vary in size from a pinhead to a small marble, while on the intestine they may be as large as an egg. The liver and spleen lesions are usually raised and are easily removed. Many of the tubercles on the intestines open into the bowel. These intestinal lesions give off countless tubercular bacilli into the intestine and thus the droppings become polluted with the disease-causing organism. While the above-listed lesions are suggestive of tuberculosis, positive diagnosis is dependent upon bacteriological examination of affected tissues.

The most important factor in the spread of avian tuberculosis is the droppings of infected birds. In lung infections, the disease may also be spread by way of mouth. Poultry yards that are contaminated with excreta of infected birds may act as a source of infection to healthy birds for several years, since the germ is highly resistant to external influences. The bacillus is, however, readily destroyed by direct sunlight. Tuberculosis may be introduced into a flock

**Fig. 18—Liver, spleen and sections of intestine taken from a fowl affected with fowl tuberculosis. Note nodules.** (After Clandinin and Robblee.)

mechanically, i.e., on the shoes of visitors, on returned shipping crates, on feed sacks, or through the purchase of infected stock.

Mortality from this disease varies considerably, depending on the degree of infection. Losses are confined chiefly to birds more than one year old.

There is no treatment for avain tuberculosis. Once the disease is detected in a flock, rigid measures must be adopted if the disease is to be brought under control. Where possible a tuberculin test should be made. If this test shows that the infection is high, the best procedure is to destroy and burn the affected birds, and sell the rest of the birds for meat purposes. Following depopulation, the houses and equipment should be thoroughly cleaned and disinfected and the ground over which the affected birds have ranged should be subjected to frequent cultivation, in order to expose the tubercular germ to the sun's rays for destruction. In connection with the disinfection of floors and equipment, water containing 3% lye will be

found inexpensive and effective. Regular spray disinfectants may be used on the walls and ceiling. Where it is found necessary to depopulate a poultry farm because of tuberculosis, no poultry should be housed in the same buildings until at least one year has elapsed and no poultry should be allowed to range over the ground previously used until it has been freed of tubercular bacilli by frequent cultivation over a period of at least one year.

Where the tuberculin test has shown that the infection is light, affected birds should be disposed of and the houses and equipment should be thoroughly cleaned. and disinfected. The remaining birds should not be permitted to run over the old range until it has been thoroughly cleaned by frequent cultivation over a period of at least one year.

Where it is not possible to have a tuberculin test made, as is the case in Alberta, the following control measures are recommended.

1. Depopulate the premises of all poultry for at least one year.
2. Thoroughly clean and disinfect the houses and equipment.
3. Clean the ground over which the birds previously ranged by cultivating it frequently during the time that the buildings are free of poultry.
4. Re-stock the farm by purchasing baby chicks, not by obtaining semi-mature or ready-to-lay stock, since by following the latter methods of re-stocking reinfection might result.
5. Dispose of each year's laying flock early enough each fall to permit thorough cleaning and disinfection of the laying quarters before the new crop of pullets are ready to be moved into the laying quarters.
6. Do not carry any yearling hens into the second laying year. They would only serve as a source of infection to the pullet flock.

To avoid mechanical introduction of tuberculosis, visitors should be discouraged from inspecting the premises, returned shipping crates should be cleaned and disinfected immediately on arrival back on the premises, contaminated feed sacks should not be used for hauling feed about the farm, and new stock should only be purchased from flocks known to be free from tuberculosis.

Since swine are highly susceptible to avian tuberculosis, farmers are cautioned against permitting their pigs to run with poultry. This precaution is particularly applicable in Alberta where tuberculosis infection in poultry is quite prevalent.

**Pullorum.**

Pullorum disease, formerly called bacillary white diarrhoea, is primarily a disease of baby chicks. It is also found in mature hens.

In the chick the disease assumes an acute, highly fatal form, whereas in the adult fowl the disease is of a chronic nature.

Pullorum disease is caused by the bacterium *Salmonella pullorum*. In the hen this bacterium often becomes localized in the ovary. Many of the eggs laid by pullorum-diseased hens are infected with the causative organism. Infected eggs, when hatched, produce infected chicks. Infected chicks pass the pullorum germ in their droppings and hence are capable of infecting other chicks, in the incubator, in shipping boxes, and in the brooder house. Many infected chicks die during the course of the disease, but some survive and grow to maturity with the infection localized in the ovary. Survivors (known as "carriers") transmit the disease through the egg to their progeny and thus the malady is perpetuated in a flock (see Fig. 19).

Pullorum disease may affect both chickens and turkeys. The disease is more or less widespread, occurring wherever poultry are kept.

Factors which predispose chicks to this disease include chilling, overheating and withholding feed.

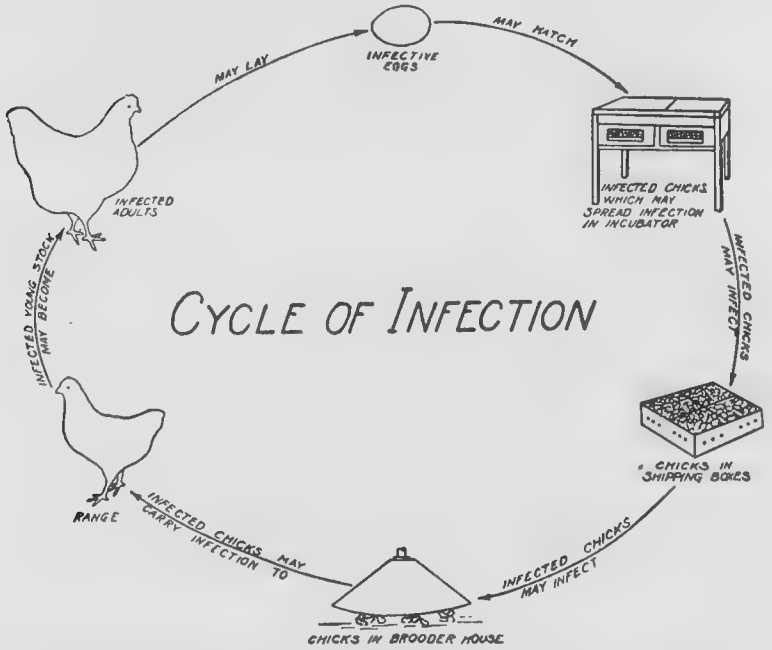



**Fig. 19—Cycle of infection of pullorum disease.** (After van Roekel.)

The symptoms of pullorum disease in baby chicks are not very definite. Some chicks die so soon after hatching that no symptoms are displayed. Others present a picture of general dejection (see Fig. 20); they stand "huddled" in corners or near the stove, with eyes closed and drooping wings and they frequently chirp plaintively, emitting shrill cries as if in pain. Diarrhoea, with "pasting-up" behind, is often evident. Death usually occurs within two or three days. Heaviest losses occur during the first ten days after hatching, but mortality may continue to be quite high up to three weeks. After this period, the disease tends to change to the chronic form. Mature fowls usually do not show any outward sign of infection.

**Fig. 20—Baby chicks showing symptoms of pullorum disease.** (After Hamilton.)

Post-mortem examination of chicks dying soon after hatching shows no significant changes. Chicks surviving for a longer time, however, may show nodules in the heart and lungs, and the presence of a large amount of unabsorbed yolk. A definite diagnosis, however, can only be made by bacteriological examination, as similar

**Fig. 21—Normal ovary taken from a laying hen.** (After Rettger.)

**Fig. 22—Pullorum infected ovary taken from a laying hen. Note misshapen yolks.** (After Rettger.)

internal changes may be caused by chilling, overheating or improper feeding.

In adult fowls the commonest post-mortem finding is misshapen, greenish or leaden-colored yolks in the ovary (see Fig. 21 and Fig. 22). In cases where the heart is involved, pericarditis (inflammation of the heart sac) and the presence of excess pericardial fluid may be evident. The heart may be enlarged (see Fig. 23) and noduled.

In chicks, mortality from pullorum varies greatly, at times approaching 100%. In adult fowls mortality from pullorum is relatively light. Pullorum disease causes decreased egg production and decreased hatchability. Economic losses from this malady are great.

The following measures are recommended in connection with the control of an outbreak of pullorum disease in chicks.

1. Kill and burn all birds showing advanced symptoms of the disease.
2. Administer sulfamethazine (Sulmet, Lederle Laboratories Inc.) in the drinking water. Use a 0.2% (1:500) concentration for two days; miss four days, give a 0.1% (1:1000) concentration for one day. and, after a further lapse of four days, give a final day of 1:1000 dilution.
3. Clean and disinfect the waterers daily.
4. Prevent contamination of the feed and water by excreta.
5. Clean the house at least every other day.

Since many of the chicks that recover from an outbreak of pullorum disease are "carriers" of the disease, birds that have gone through a seige of the disease should be sold as broilers.

**Fig. 23—Pullorum disease in adult bird. Right, normal heart; left infected heart showing pericarditis (inflammation of heart-sac) and enlargement of heart.** (After Biely).

Now while it is true that pullorum disease in chicks may be controlled by following plans such as the one just outlined, prevention of losses from this disease involves: the identification of infected birds by a reliable pullorum test, the removal of infected birds from the breeding flock, the cleaning and disinfection of the premises, and the observation of rigid measures of sanitation and disinfection during incubation.

There are at least three reliable methods of testing adult birds for pullorum disease: (1) the tube-agglutination test, (2) the rapid-plate serum test, (3) the rapid whole-blood stained-antigen test. The tube-agglutination test and the rapid-plate serum test are conducted in the laboratory, a sample of blood being taken at the farm from each bird on the premises and shipped to the laboratory where the test is performed. The band numbers of all reacting birds are sent to the flock owner, who is then able to sort out and dispose of his "carriers". The rapid whole-blood stained-antigen test (see Fig. 24) is conducted at the farm and incidently is the test employed in Alberta for the detection of "carriers" in the hatching egg supply

flocks. This test has marked advantages over the two previously referred to tests in that; (1) it is considerably less expensive, (2) it is better adapted to application under conditions where shipping distances to the central laboratories are great, and (3) in that it necessitates only one handling of the fowls, since it is possible to remove reacting birds at the time the test is being conducted. While no one of these three tests is considered 100% accurate in detecting "carriers", nevertheless, from the standpoint of reducing losses caused by pullorum disease, they are of inestimable value. The consensus at the present time as to the comparative accuracy of the test when used on chickens is that they are equal when conducted by qualified personnel.

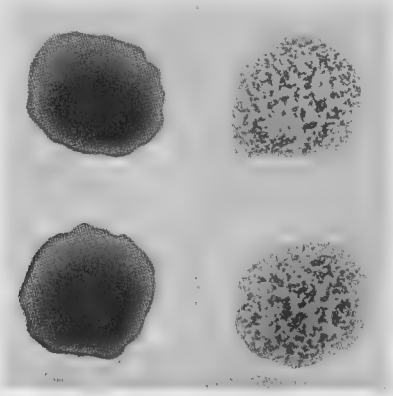


**Fig. 24—Rapid whole-blood stained-antigen test for pullorum disease. Left. negative reaction; right, positive reaction.** (After Biely and Moynihan.)

Pullorum disease, as previously indicated (see Fig. 19) is readily spread from one chick to another at hatching time. This is especially true in forced-draft incubators. In order to arrest this mode of spread of infection, incubators should be fumigated.

Formaldehyde has been found very effective in incubator disinfection. Briefly the procedure is as follows: Compute the number of hundred cubic feet of air space in the incubator. Next, calculate the quantities of formalin and potassium permanganate needed on the basis of 140 cubic centimeters formalin (4 2/3 oz.) and 70 grams (2 1/2 oz.) potassium permanganate for each one hundred cubic feet of air space to be fumigated. Now place the measured amount of potassium permanganate in an enamel pan or pyrex dish large enough to hold about ten times the quantity of formalin to be used. Set this pan in the bottom of the incubator. For large incubators more than one pan may be necessary. Next, pour the computed amount of formalin over the permanganate crystals and

close the incubator door as quickly as possible. Leave the incubator door closed for fifteen minutes. At the end of this time air out the machine. Excess formaldehyde may be neutralized by placing a small dish of ammonium hydroxide under one of the fans or by sprinkling ammonia water on the inside walls of the incubator.

In connection with formaldehyde fumigation it is recommended that the fumigation be done on the 19th day of incubation or just before "pipping" has commenced. During the fumigation period humidity in the incubator should be maintained at 70%. Operators are cautioned against fumigating machines that contain eggs which have undergone only 24 to 72 hours of incubation. Embryos at this age may be injured by formaldehyde fumigation.

Poultrymen who desire to eradicate pullorum disease from their flocks and who wish to keep their flocks free of pullorum disease should:

(1) Eliminate "carrier" birds by blood testing (the blood testing work in Alberta is done by the Poultry Branch, Provincial Department of Agriculture, Edmonton). The first test should be done shortly after the pullets come into lay. If any reactors are detected they should be disposed of immediately and the remaining birds should be retested at thirty-day intervals until such time as two consecutive negative tests are obtained. Once the flock has been shown to be free of pullorum reactors on two consecutive tests, testing at twelve-month intervals is all that is necessary. Following the removal of each lot of reactors, the premises should be thoroughly cleaned and disinfected.

(2) Purchase eggs for hatching baby chicks and new stock only from sources that are known to be free from pullorum infection.

(3) Maintain sanitary conditions on the farm, particularly in the incubator and in the quarters used for brooding and rearing young stock.

**Fowl Typhoid.**

This is an infectious disease. In young stock the disease is acute in nature, whereas in adult stock both acute and chronic forms are known to occur.

Fowl typhoid is caused by the micro-organism *Shigella gallinarum*. In an infected hen this bacterium is often present in the ovary. The disease is transmitted from hen to chick through the egg in a similar manner to that referred to in the discussion on pullorum disease (see Fig. 19).

Both chickens and turkeys are susceptible to fowl typhoid and the disease is more or less widespread in occurrence. The disease is encountered most commonly in late summer.

The principal symptoms of fowl typhoid are: loss of appetite, sleepiness, droopiness, paleness about the head, yellowish or greenish diarrhoea and high fever. In the acute form the disease runs its course in from 2 to 10 days.

Post-mortem examination of birds affected with the acute type of the disease reveals a greenish bronze colored liver, watery blood, enlarged spleen and kidneys and frequently grayish spots on the liver and heart. In the chronic form, in addition to these changes, ovaries similar to those infected with pullorum disease (see Fig. 21 and Fig. 22) and the presence of serous fluid in the pericardium (heart sac) and the body cavity are often noted. Positive diagnosis of fowl typhoid rests on bacteriological examination of affected birds.

The droppings of infected birds contain large numbers of the typhoid organism and hence serve as a source of infection for healthy birds.

Mortality from this disease varies greatly. In some instances very few losses are sustained, while in other instances entire flocks are wiped out.

Control of an outbreak of typhoid may be effected by following the steps outlined for the control of an outbreak of pullorum disease in chicks (see Pullorum).

In flocks where fowl typhoid has been detected, steps should be taken to eradicate the malady. A pullorum test (which also detects birds harbouring the typhoid organism) should be conducted at monthly intervals until two consecutive tests show no reactors. All reactors should be disposed of as soon as detected. Following each test in which reactors are found, the houses and equipment should be thoroughly cleaned and disinfected. Under no circumstances should the non-reacting birds be permitted to run over their old range, for this would only result in further infection. The old range should be cleaned up by cultivating frequently, so as to expose the typhoid germ to the elements for destruction. One season's cultivation should be sufficient.

**Paratyphoid.**

Paratyphoid infections in chickens and turkeys are of considerable importance to the poultry industry. Over fifty types of Salmonella have been recovered from birds affected with paratyphoid.

The disease is quite prevalent in Alberta and in other parts of the Dominion. Young birds are more susceptible than adult birds.

Dullness, listlessness, weakness, sagging wings and diarrhoea are symptoms frequently noted in chicks affected with paratyphoid. Loss of appetite, unthrift, emaciation, unkempt appearance and diarrhoea, on the other hand, are symptoms of the disease in adult birds. Internal findings in paratyphoid are not constant. In some instances enlargement of the liver and spleen, cheesy plugs in the caeca (blind pouches of the intestine), inflammation of the duodenum (U-shaped part of the small intestine) and/or lower portion of the small intestine may be noted. Diagnosis of paratyphoid is difficult and is dependent upon the isolation and identification of Salmonella organisms, other than the two (*S. pullorum* and *S. gallinarum*) that cause pullorum disease and fowl typhoid, from the internal organs and/or intestinal contents.

Paratyphoid is believed to be transmitted from hen to chick through the egg in much the same manner as pullorum disease (see Pullorum). The causative organisms are also passed in the droppings of affected birds.

Losses resulting from an outbreak of paratyphoid in chicks or poults may be minimized by following the program outlined for the treatment of an outbreak of pullorum disease in chicks (see Pullorum). Birds that have recovered from paratyphoid should not be retained for breeding purposes since many would be "carriers" of the disease.

Control measures for paratyphoid are similar to those outlined for pullorum (see Pullorum) except that in the case of paratyphoid the antigen used for the detection of adult "carriers" must correspond to the paratyphoid organism present in the flock.

**Fowl Cholera.**

Fowl cholera is an acute infectious disease. Occasionally it occurs as a chronic disease although it is generally characterized by sudden death and high mortality.

The disease is caused by the micro-organism *Pasteurella multocida*. In infected birds this organism is found in large numbers in the blood and in various organs of the body. It produces a septicemia, that is, a blood poisoning.

Fowl cholera is widespread in occurrence and is known to affect both chickens and turkeys. The disease is encountered most frequently during the winter months.

The symptoms of this disease vary widely. The onset is usually sudden. Often the first indication of the presence of this disease is that birds are found dead in the nests or under the roosts. As the outbreak progresses the disease assumes a less acute form. In the less acute form, the symptoms commonly noted are: loss

of appetite, droopiness, sleepiness, darkness about the head, yellowish or greenish diarrhoea, restrained breathing, high fever, increased thirst and oedema of the wattles.

Post-mortem examination of birds that die suddenly from fowl cholera often shows no internal changes. However, similar examination of birds that have been affected for several days with this disease reveals: accumulation of sticky mucus in the mouth, congestion of the visceral organs, pin-head size necrotic areas in the liver, friable liver, haemorrhages of various sizes in the heart, gizzard and mesenteries (see Fig. 25), straw-coloured fluid in the pericardium (heart sac), inflammation and pin-point haemorrhages

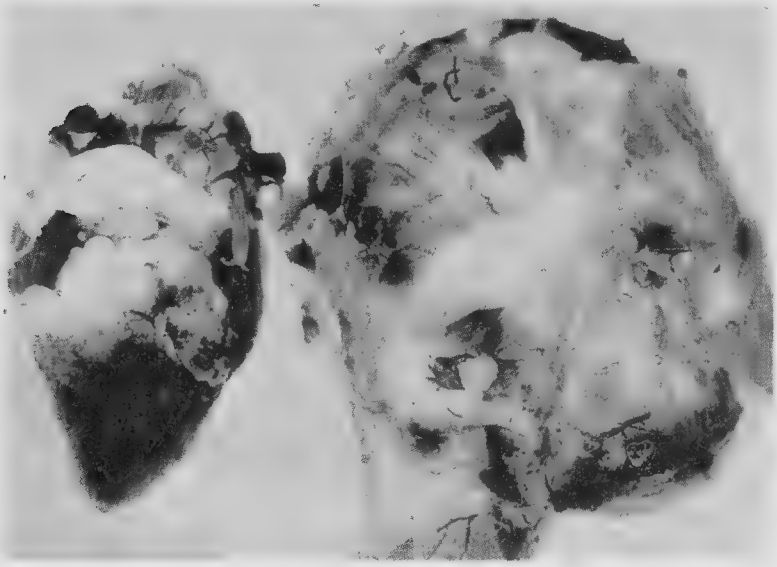


**Fig. 25—Haemorrhages on heart, gizzard and proventriculus in fowl cholera. (After Biester.)**

in the upper portion of the intestine and cheesy yellow deposits among the intestines. In the chronic form, cholera has been known to cause swollen joints, bumblefoot, peritonitis (inflammation of membrane covering the viscera) and ascites (accumulation of serous fluid in the abdominal cavity). Positive diagnosis of fowl cholera is dependent upon bacteriological examination of the blood of infected birds.

As with fowl tuberculosis and fowl typhoid, the droppings from infected birds constitute the most important factor in the spread of this disease. The germ causing the disease gains entrance to the bird's body by way of the respiratory and digestive tracts.

In the acute form this disease may practically wipe out a flock in the course of a week or two. - Losses from chronic cholera are light.

The following measures are recommended in connection with the control of an outbreak of fowl cholera.

1. Kill and burn all birds showing advanced symptoms of the disease.
2. Administer sulfaquinoxaline (Sulfaquinoxaline sodium in excess sodium hydroxide, Merck and Co. Ltd.) or sulfamethazine (Sulmet, Lederle Laboratories Inc.) in the drinking water. Use a 0.025% (1:4000) concentration of the former administering same on a two day on, three day off, two day on, three day off, two day on basis or use a 0.2% (1:500) concentration of the latter for two days, miss four days and use a 0.1% (1:1000) concentration for one day, and after a lapse of four more days give a final day of 0.1% concentration.
3. Clean and disinfect the waterers daily.
4. Prevent contamination of the feed and water by excreta.
5. Thoroughly clean and disinfect the house and equipment.

Since ground over which infected birds have ranged constitutes a reservoir of infection it should not be used again for poultry until it has been cleaned up by frequent cultivation over a period of at least one year.

**Erysipelas.**

Erysipelas is a disease that affects both chickens and turkeys although it is more frequently noted in the latter. It is caused by the micro-organism of swine erysipelas, *Erysipelothrix rhusiopathiae.*

The disease is usually found in birds that are permitted to range with swine that are affected with the disease or in birds that are allowed to range over ground previously contaminated by infected swine.

Most outbreaks in turkeys occur during the fall. Losses are heaviest amongst males. Symptoms include general weakness, listlessness, loss of appetite and passage of yellowish or greenish diarrhoea. Sick birds tend to crouch with lowered heads. Their heads often appear bluish in color. In the male, the snood may be quite swollen (see Fig. 26). Internal examination reveals blotchy haemorrhages in the superficial muscles, thick mucus in the nasal passage, enlargement and congestion of the liver, spleen and kidneys and the presence of catarrhal exudate in the intestine. Diagnosis should be based on the isolation and identification of the causative agent from the tissues of affected birds.

**Fig. 26—Erysipelas. Note swollen snood.** (After Rosenwald and Dickenson.)

In the control of an outbreak of erysipelas the following measures are recommended:

1. Separate sickly birds from apparently healthy birds.
2. Inject 100,000 units of procaine penicillin intramuscularly into each affected bird. (Recently it has been shown that N,N dibenzlethylenediamine dipenicillin G, 200,000 units per bird, is also highly effective against *E. rhusiopathiae.*)
3. Move sickly and apparently healthy birds to separate clean ranges.
4. Inject any birds in the apparently healthy group, that become sick, with penicillin and transfer to the other group.
5. Disinfect the waterers daily.

**Coryza.**

The terms, cold, roup and coryza have been applied by poultrymen more or less interchangeably in referring to inflammations of the mucous membranes of the upper respiratory tract. Coryza, however, is perhaps the proper designation for a simple cold. The consensus at the present time as to the cause of coryza is that *Hemophilus gallinarum* is capable of producing the condition, and that several other agents play important roles in the production of symptoms.

The first birds to contract coryza are those of low vitality. Hence any factors that tend to affect vitality adversely must be considered as predisposing to coryza. Overcrowding, improper ventilation, improper feeding, lack of vitamin A, infestation with lice, mites, worms or coccidia, and undue exposure to inclement weather, drafts, dampness or unsanitary conditions, all fall in this category and should therefore, be avoided.

Early stages of coryza are characterized by watery eyes (see Fig. 27) and a serous discharge from the nostrils. As the disease progresses, the nasal discharge becomes thick, sticky and foul smelling (probably the result of secondary infection), and tends to form yellowish crusts about the nasal openings. With the extension of the inflammation, the sinuses become filled with mucus (see Fig. 27) which, being unable to drain away, changes to a cheesy material and causes the sinuses to bulge. Similar cheesy material also accumulates in the cleft in the roof of the mouth. In very advanced cases, cheesy patches are often found in the mouth itself. These cheesy patches in the mouth are readily distinguished from those noted in the diphtheritic form of fowl pox in that they are not so adherent as those caused by fowl pox. In cases where the trachea (windpipe) is involved, difficulty in breathing is experienced. This is caused by an accumulation of mucus in the trachea. Severely affected birds usually sit quietly, with feathers ruffled, show little or no inclination to eat, wheeze and sneeze at regular intervals, and frequently shake their heads in an effort to dislodge mucus.

**Fig. 27—Watery eye and swollen sinus in a fowl affected with coryza.**
(After Iowa Agricultural Experiment Station.)

Post-mortem examination shows no characteristic lesions other than those described above.

Coryza is one of the most common ailments affecting poultry. While mortality losses from this disorder are of a relatively low order, losses resulting from poor growth in growing birds and poor egg production in laying birds are very great.

Preventive measures involve giving due consideration to all factors that may have deleterious effects on vitality and so predispose to coryza. Overcrowding should be avoided. Lightweight layers should have three square feet of floor space, heavyweights four. Poultry houses should be properly ventilated. Openings should be so arranged as to permit a steady circulation of air through the house, the moist, foul air being drawn off and replaced by fresh air. Drafts should be avoided, especially is this true of drafts over the roosts. Birds should not be unduly exposed to inclement weather, and should be kept free of lice, mites, worms and coccidia, for all tend to lower vitality and predispose to disease. Further, care should be taken to see that the ration used is well balanced and that it is properly administered.

Even under the best of management, colds may develop. Accordingly one should watch his birds very closely. On the slightest sign of infection, the predisposing causes should be determined and corrected.

As far as success in treating affected birds is concerned, much depends on the stage which the disease has reached. Mild cases are readily cleared up by administering a 0.1% concentration of sulfamethazine (Sulmet, Lederle Laboratories Inc.) in the drinking water for a period of two to three days or by intramuscular injection of 0.2 grams of dihydrostreptomycin sulphate into each bird. Advanced cases require individual treatment similar to that referred to under sinusitis in turkeys (see Chronic Respiratory Disease).

**Chronic Respiratory Disease.**

Chronic respiratory disease, commonly referred to as CRD, is a disease that affects both chickens and turkeys. In chicks, in addition to the terminology CRD the disorder is frequently called "air sac infection" while in turkeys the disease is generally known as "sinusitis". The same agents are, however, considered to be responsible for the disease in chickens and turkeys. Opinion is that two agents are involved, one a virus and the other, so-called, pleuropneumonia-like organisms (PPLO). It also appears probable that other secondary agents produce wide variation in the form, course and severity of the disease.

*Chickens.*—In chickens CRD or air sac infection is frequently noted in stock originating from hatching egg supply flocks of questionable CRD history. This is because of the fact that the disease is transmitted through the egg. The disease may appear at any age but causes highest mortality in birds six to eight weeks old.

With the onset of the disease there is usually a marked drop in feed consumption. Affected birds shake their heads and develop

a hacking cough which is particularly evident at night. As the disease progresses a continuous breathing rattle develops and marked gasping for breath occurs. The eyes may water and there may be a mucous discharge from the nostrils. As the disease enters the chronic stage the respiratory symptoms generally subside and the birds appear listless showing little interest in eating. As a result further weakening and loss of weight occurs. In young chickens, at this stage, death losses begin to mount. In some instances mortality of the order of 30% is experienced; however, in most instances mortality is in the one to ten percent range. In addition a high percentage of culls results from the infection. In laying birds not only are death losses serious but there is a gradual and prolonged drop in egg production. If nothing is done to correct the situation the birds may remain listless for weeks on end.

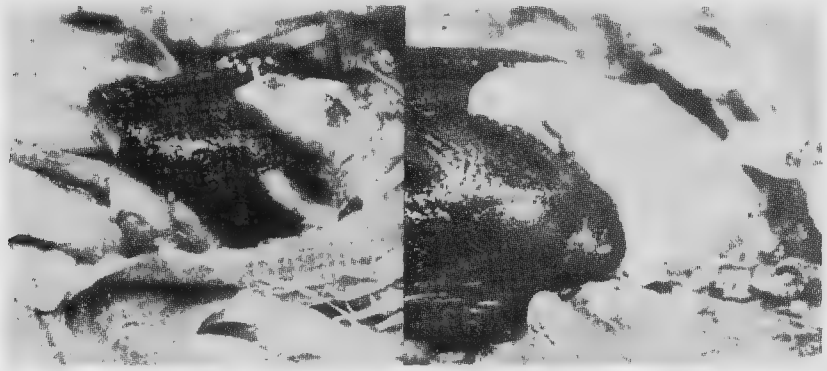


**Fig. 28—Chronic respiratory disease. Note slimy exudate on the liver, and enlarged and thickened heart sac.** (After Dr. Salsbury's Laboratories.)

On internal examination, in early stages of the disease, frothy mucus may be noted in the air sacs. As the disease progresses exudate may be found in the trachea, bronchial tubes and in the thoracic and abdominal air sacs. The air sacs may appear cloudy or yellowish. Later an increased amount of caseous material may be found covering the walls of the air sacs and sometimes filling the air sacs. The liver may be covered with a slimy, yellowish-gray false membrane (see Fig. 28) and the pericardial sac (heart sac) may be considerably thickened (see Fig. 28). Cheesy material may also be found in the body cavity.

*Turkeys.*—In turkeys the disease may affect the lower respiratory tract or may be confined to the head region. In the former case the external and internal symptoms are similar to those described above for air sac infection in chickens and mortality rates as high as 25% may result. Where the disease is localized in the head

region (sinusitis) the first symptoms include watery eyes and a nasal discharge. Affected birds sneeze, cough and breath with considerable difficulty. The birds frequently shake their heads to dislodge mucus from their nostrils and attempt to wipe the discharge on their wing feathers. As the disease progresses mucus collects in the sinuses causing them to bulge (see Fig. 29). Initially the mucus in the sinuses is quite liquid; however, it soon becomes caseated.

**Fig. 29—Head of turkey affected with sinusitis. Note swelling under the eye.** (After General Mills.)

In arriving at a diagnosis, account should be taken, not only of the above referred to external and internal symptoms, but also of the fact that CRD is not generally characterized by rapid spread and short duration with excessive death rates as is typical of the virus-caused respiratory diseases (Newcastle Disease, Laryngotracheitis and Bronchitis). For a positive diagnosis it is usually necessary to have a PPLO-HI test conducted at a diagnostic laboratory. This test takes into account the fact that the organism that causes CRD has the ability to agglutinate chicken red blood cells. Hence, serum from an infected bird should contain antibodies to prevent this phenomenon from happening. If blood serum from a bird does the latter, it would indicate that the bird is infected with CRD.

Egg transmission of CRD has been widely accepted as the main means of dissemination of this disease. In this connection it has been shown that both the PPLO and the virus may be transmitted through the egg. The egg transmission rate, however, is low. Transfer of the disease from bird to bird by direct contact is recognized. The agents responsible for the disease are also transmitted through the air, a fact that should be taken into account when

brooding chicks from different sources in one pen or when introducing new stock onto a premises which contains birds proven free of PPLO.

Chronic respiratory disease is one of the most serious diseases with which poultrymen have to contend. In broiler flocks it not only can be responsible for appreciable mortality but can decrease the growth rate and feed efficiency to such an extent that it may be unprofitable to raise broilers. In laying and breeding flocks, decreased egg production and poor hatchability account for the main economic losses from CRD infection. The financial loss resulting from actual deaths and loss of condition in adult birds, of course, should not be overlooked.

Treatment of chicks affected with CRD usually involves the inclusion in the ration of high levels of aureomycin (200 to 400 grams per ton of ration), terramycin or streptomycin (150 grams per ton of ration) or a combination of streptomycin and penicillin (75 grams streptomycin and 25 grams penicillin per ton of ration). As a preventive, some flock owners follow the practice of including a moderately high level of antibiotic (50 grams per ton of feed) in their starter or broiler feeds on a continuous basis. The economics of this practice, however, should be studied before adopting same. It, perhaps, should be pointed out here that it is generally considered that antibiotics in the feed produce their beneficial effect by preventing infection by secondary invaders rather than by destroying the CRD agents.

Outbreaks of sinusitis in turkeys have been brought under control by incorporating streptomycin and penicillin in the feed at the rate of 75 grams of streptomycin and 25 grams of penicillin per ton of ration. Individual treatment is also recommended. The latter involves draining the sinuses (when filled with liquid) by means of a 10 or 20 cubic centimeter syringe with a 16 gauge, 1½ inch needle, and introducing into the drained sinuses, by means of another syringe, 5 mg. of terramycin in mineral oil, 100 mg. of terramycin in 0.9% saline, 1 cc. of 4% silver nitrate solution or 1 cc. of 10 to 15% argyrol.

While the above referred to treatments for CRD in chicks and sinusitis in turkeys may prove of some value in treating birds affected with the disease, they contribute little to the overall control of the malady. Unfortunately too, because of the chronic nature of CRD, there appears to be very little likelihood of controlling this disease through vaccination. Insofar as control and prevention of CRD in chickens and sinusitis in turkeys is concerned it would appear that PPLO-free breeding flocks should be established. To do this, breeding birds should be checked for the presence of PPLO in-

fection by using the PPLO-HI test. If the infection is low, infected birds should be eliminated and PPLO-free eggs obtained for hatching purposes from the remaining birds. If, however, the infection is extensive it would likely be economically unsound to discard all infected birds. In order to establish a PPLO-free breeding flock from such an infected flock, all of the breeders in the infected flock should be individually injected with 150 to 200 mg. of streptomycin and 150,000 units of penicillin. This treatment will reduce the PPLO egg transmission rate during the following three or four weeks to a very low level. Further, to prevent mechanical transfer of infection, the hatching eggs should be dipped in germicidal solution and the eggs should be hatched in incubators containing eggs that come only from PPLO-free flocks. The chicks, so obtained, should be reared in isolated groups so that if CRD should break out in any one group that group may be discarded before infection is transferred to the other PPLO-free groups. In addition the young chicks should be vaccinated against infectious bronchitis (and Newcastle disease in areas where it is a problem) since chicks that are not protected against these two diseases seem more prone to develop CRD than vaccinated chicks. Once a PPLO-free flock is established no stock introductions except from CRD-free sources should be considered. The continued freedom of the flock from PPLO infection then becomes just a matter of periodic checking by the PPLO-HI test to see that the flock is free of PPLO infected birds keeping in mind that if "carriers" are detected, the above irradication procedure must again be implemented.

**Infectious Synovitis.**

Recently a condition known as infectious synovitis or infectious anemia-synovitis has been described as affecting broilers of six to eight weeks of age causing serious mortality losses. Evidence available, would appear to point to a small bacterium or large particle virus as the cause of the disease.

Initial symptoms include paleness about the head and leg weakness. Affected birds assume a sitting position (see Fig. 30) and when forced to walk, the gait is unsteady and stilted. As the disease progresses, the feathers become ruffled particularly in the head and neck region and the combs of the males tend to shrivel and become pale or purplish-red in color. The hock joints frequently swell and breast blisters are not uncommon. Affected birds become quite thin in spite of the fact that they continue to eat until shortly before death. A greenish colored diarrhoea usually is present.

On post-mortem examination, affected birds appear dehydrated, the spleen is frequently enlarged and the bursa (pouch off the large intestine just inside the anus or vent) is found enlarged and filled

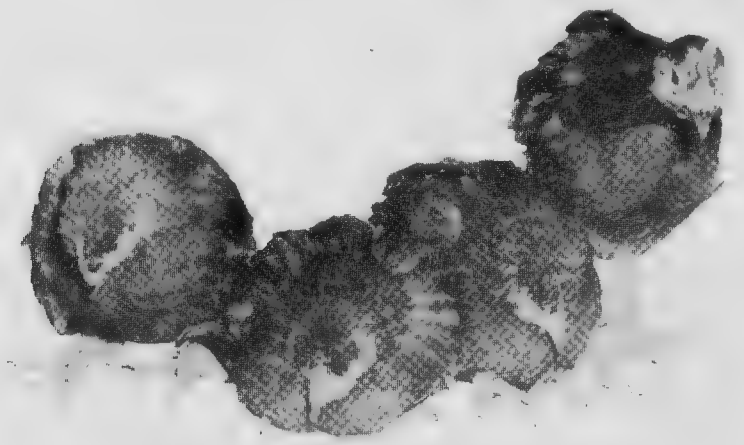


**Fig. 30—Infectious synovitis. Note leg weakness and shrivelled comb.**
(After West Virginia University Agricultural Experiment Station.)

with caseous exudate. Upon opening the hock swellings a creamy exudate is found. In the later stages of the disease this exudate takes on a cheesy consistency. A study of the blood of affected birds reveals reduced hemoglobin content which can be accounted for on the basis of reduced number of red blood cells and increased number of white blood cells. Diagnosis of synovitis is usually based on the aforementioned symptoms and post-mortem findings.

The manner by which this disease is spread from one bird to another or from one farm to another is not understood. Transmission in the laboratory has been effected by inoculation of joint fluid into the foot pad, hock joint, sinus or air sac.

This disease has been responsible for serious financial loss to broiler raisers. High mortality, increased number of culls and decreased feed efficiency have all contributed to this loss.

Trouble from synovitis may be avoided by feeding, on a continuous basis, a ration containing 50 to 100 gm. of furazolidone (Dr. Hess and Clark, Inc.) per ton of feed. In addition continuous high level feeding of aureomycin (200 gm. per ton of ration) or of furazolidone (100 to 200 gm. per ton of ration) appears to curtail spread of the malady in affected flocks and in some instances has proven helpful in bringing outbreaks under control. Intramuscular injection of affected birds with 200 mg. dihydrostreptomycin sulphate also shows promise as a control measure for this disease.

**Omphalitis.**

Omphalitis, or navel ill, is a disease of chicks and poults in which the navel appears abnormal. It is the result of bacterial infection

of the navel at hatching time. A number of different micro-organisms are capable of causing the condition. The abnormality is oftimes encountered in chicks and poults that have been hatched under unsanitary conditions.

The principal symptoms of the disorder are: pus about the navel, puffiness in the abdomen (see Fig. 31), and the presence of diarrhoea. Internal examination reveals: putrefaction about the navel, unabsorbed yolk, yellowish liver, and distention of the gall bladder.

**Fig. 31—Omphalitis. Left, normal chick. Right, chick affected wtih omphalitis in a severe degree.** (After General Mills.)

The disease shows up the second or third day after hatching. Heaviest losses occur between the third and sixth day after hatching. There is no effective treatment for this disorder. It may, however, be prevented by observing proper incubator sanitation. This entails thoroughly cleaning and disinfecting the incubator before the hatching season commences and fumigating the incubator with formaldehyde gas before each hatch. For details on incubator fumigation see Pullorum disease.

**Botulism.**

Botulism, also known as limberneck, is an acute or sub-acute intoxication caused by exotoxins (poisonous substances secreted or excreted by bacteria), produced by the microscopic *Clostridium botulinum*, types A and C. The organisms themselves do not affect the fowl adversely, but the exotoxins produced by them during the process of their multiplication in decaying matter do. Minute amounts of the toxins (types A and C) are sufficient to produce severe toxic effects and only slightly more is lethal.

Outbreaks of botulism occur chiefly during warm weather, as it is during such weather that putrefaction is going on most rapidly. Both chickens and turkeys are susceptible to botulism.

The principal signs of botulism are: sleepiness, limpness of neck and wings (see Fig. 32), incoordination, prostration, and looseness of the feathers. Post-mortem examination reveals no changes in the organs. The symptoms displayed by affected fowls are the chief means of diagnosing botulism.

**Fig. 32—Botulism. Note paralysis of neck.** (After Hamilton.)

Treatment of badly affected birds is of little avail. However, Epsom salts given at the rate of one teaspoonful per bird (two pounds per hundred birds), or castor oil at the rate of one-half ounce per bird will aid in flushing the toxins out of the intestinal tract of less severely affected birds, and as a consequence effect their recovery. Following the purge, the houses should be cleaned so as to remove toxin eliminated into the litter. To prevent further spread or recurrence of the trouble, the source of the toxins must be found and removed. In this connection, the premises should be carefully searched for decomposing organic material, which as previously stated, constitutes the source from which birds obtain the toxins.

## PROTOZOAN DISEASES

Protozoan diseases are diseases caused by minute organisms called protozoa. (Protozoa are one-celled organisms, the smallest in the animal kingdom.) The more important diseases in this group are coccidiosis, blackhead, hexamitiasis and trichomoniasis.

**Coccidiosis.**

Coccidiosis is an intestinal disease caused by microscopic parasites known as coccidia. In young birds the disease is usually acute in nature, while in old birds the disease generally follows a chronic course.

Eight species of coccidia are known to infect the domestic fowl. They are: *Eimeria tenella, E. necatrix, E. mitis, E. acervulina, E. praecox, E. maxima, E. hagani* and *E. brunetti.* Generally speaking, only two of these species are decidedly pathogenic: *E. tenella* affects the caeca (blind pouches of the intestine) of chicks, causing acute coccidiosis; *E. necatrix* affects the duodenum (U-shaped part of the small intestine) and mid-portion of the small intestine of fowls of four months of age and over, causing, in some instances, acute coccidiosis, but more often chronic coccidiosis. At least seven species of coccidia, *E. meleagridis, E. meleagrimitis, E. dispersa, E. gallopavonis, E. adenoeidis, E. innocua* and *E. subrotunda* produce the disease in the turkey.

Large numbers of coccidia are passed in the droppings of infected birds. The organisms are voided in the stage of their life cycle known as "oocysts" (see Fig. 33). Oocysts are not directly infective to birds, but, under favourable conditions of warmth and moisture, they undergo a change (sporulation) in twenty-four to seventy-two hours, giving rise to what are known as "sporulated oocysts" (see Fig. 33). Sporulated oocysts are highly infective to susceptible birds. Upon being ingested by a susceptible bird, they (sporulated oocysts) give rise in the intestine to small banana-shaped forms called "sporozoites". The sporozoites penetrate the cells lining the intestine or caeca (depending on the species) and give rise to forms known as "schizonts". The schizonts undergo a series of nuclear divisions (schizogony) and eventually split into a number of nucleated fragments or "merozoites". These merozoites, upon release, invade other cells to give rise to more schizonts, which, in turn, undergo schizogony to give rise to more merozoites. This schizont to merozoite to schizont development is repeated several times (the number of times varies with the species). Then a change occurs. Instead of more schizonts being formed from merozoites, male and female forms are produced. These, upon release into the intestinal tract, unite to form oocysts, which, as stated earlier, are voided in the droppings and are not infective until sporulated by the action of heat and moisture. Under average conditions, the species of coccidia infecting the domestic fowl and turkey take from four to ten days to complete their life cycle.

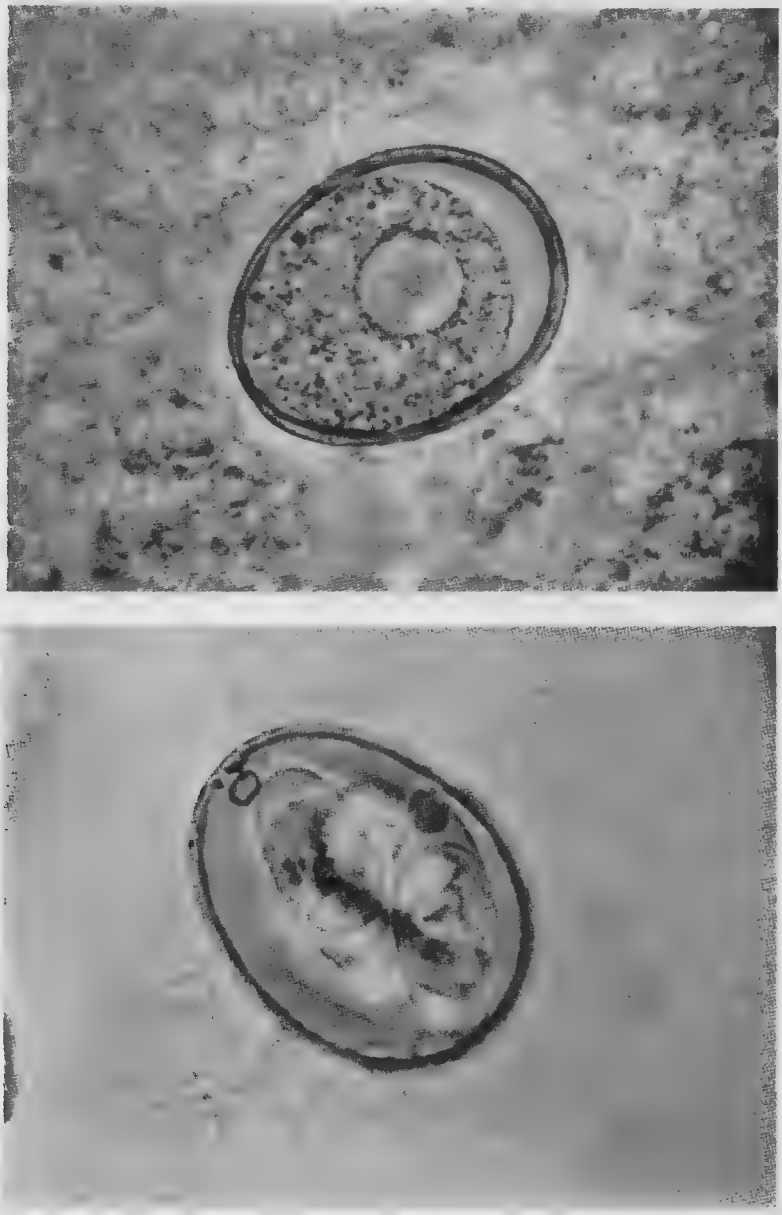


**Fig. 33—Coccidiosis. Oocyst of coccidium parasite. Top, as it appears in fresh droppings; bottom, after sporulation. Greatly enlarged.** (After Beach.)

It is of interest to note here that each intestinal cell invaded by a sporozoite, or by a merozoite, is destroyed during the course of the development that ensues. Where extensive destruction of cells has occurred the absorptive area of the intestine is greatly reduced, and as a result absorption of nutrients is impaired. Furthermore where a large number of cells in one area are destroyed, haemorrhage of blood vessels in that area takes place with the loss of much blood into the intestine.

Coccidiosis is of widespread occurrence. The incidence of this disease is greatest during the spring and summer months. This, of course, is because of the fact that during those months, conditions are ideal for the sporulation of dormant oocysts. Chickens and turkeys of three to twelve weeks of age are highly susceptible to coccidiosis. In chickens and turkeys of this age, the disease is acute in form, and causes heavy losses. Older chickens and turkeys may also be affected with coccidiosis. However, in older birds, the disease is usually chronic in form, although acute outbreaks occasionally do occur.

The principal symptoms noted in outbreaks of acute coccidiosis are: loss of appetite, emaciation, weakness, droopiness and passage of blood in the droppings. Chronic coccidiosis is manifested by: loss of appetite, progressive emaciation, sluggishness, paleness and leg weakness.

Post-mortem examination of birds afflicted with acute coccidiosis shows enlargement of caeca, thickening and inflammation of the walls of the caeca (see Fig. 34) and the presence of blood in the caeca (see Fig. 35). In cases where the disease has been less acute,

**Fig. 34—Coccidiosis. Note enlargement of the caeca and bloody splotches or areas of congestion.** (After Illinois Agricultural Experiment Station.)

cheesy blood-stained masses are often found in the caeca. Where the disease has run a chronic course, changes are usually confined to the duodenum and mesenteric part of the small intestine. The intestinal walls are thickened and inflamed and small haemorrhages are often noted. Positive diagnosis of coccidiosis is dependent upon the finding of coccidia in intestinal scrapings. This, of course, necessitates the use of a microscope.

The droppings of infected birds are the chief source of infection to other birds. Ground, houses and equipment contaminated with faeces from infected birds may serve as a source of infection to susceptible birds for as long as two years. Birds that have recovered from the disease, and birds afflicted with the disease in chronic form, are a constant menace to the well-being of other birds in the flock, for they are continually passing oocysts in their droppings. The disease may be introduced into a flock through the purchase of infected birds or by permitting birds in the flock to have access to contaminated ground, houses or equipment.

Mortality from coccidiosis is often quite high, especially where control measures are not instituted promptly. Losses in young chicks and poults usually come all at one time, whereas losses in adult chickens and turkeys are generally spread over a longer period.

**Fig. 35—Acute coccidiosis. Note blood in the incised caecum.** (After Illinois Agricultural Experiment Station.)

An outbreak of acute coccidiosis in young or adult chickens or turkeys may be brought under control by carrying out the following program. Confine all affected birds to a house for a period of ten days. Change the litter daily during the confinement period. This is necessary in order to prevent the birds from coming into contact with oocysts that have developed to the infective stage. See that the feed and water are not contaminated with fecal material.

Keep the birds away from the damp, dropping-polluted litter about the water fountain by placing the fountain on a wire stand. If reinfection has not occurred by the tenth day, all the coccidia in the birds will have been voided as oocysts and the disease will have been brought under control.

An alternative and more up-to-date treatment program consists of administering sulfamethazine (Sulmet, Lederle Laboratories Inc.) or sulfaquinoxaline (Sulfaquinoxaline sodium in excess sodium hydroxide, Merck and Co. Ltd.) in the drinking water. Treatment should commence as soon as possible after infection has been detected. The former is added to the drinking water at a 0.1% (1:1000) concentration and is administered on a two day on treatment, four day off, one day on, four day off and one day on treatment basis. The latter sulfa drug (sulfaquinoxaline) is used at a 0.025 (1:4000) concentration in the drinking water on a two day on treatment, three day off, two day on, three day off, two day on treatment basis. During the period that the birds are receiving medicated drinking water the litter in the house should not be changed since contact with the infective agent during medication is essential to the development of immunity to further infection.

After the confinement or sulfa treatment period has elapsed, the birds should be moved to clean range, that is, to range to which poultry have not had access during the previous two years. The birds should not be permitted to run over their old range, for this might result in a new outbreak of the disease.

The contaminated range may be cleaned up by cultivating. No poultry should be permitted on the old range until at least two years time has elapsed.

On broiler plants or on farms where coccidiosis is a constant problem, losses from acute coccidiosis may be minimized by adding Nicarbazin (a molar complex of 4, 4'-dinitrocarbanilide and 2-hydroxy-4, 6 dimethylpyrimidine, Merck and Co. Ltd.), sulfaquinoxaline powder (Merck and Co. Ltd.) to starting and growing rations at the rate of 1/4 lb. per ton of feed or by adding Megasul 25% Nitrophenide (Lederle Laboratories Inc.) or NFZ-Mix (11.2% nitrofurazone, Dr. Hess and Clark Inc.) to starting and growing rations at the rate of 1 lb. per ton of feed.

Chronic coccidiosis is best controlled by frequent culling of sickly birds, and by the observation of proper sanitation on the farm.

In general, losses from coccidiosis may be prevented by:

(1) Rearing young stock on clean range away from old stock.

(2) Observing proper sanitation on the farm, that is, cleaning the dropping boards, houses and yards regularly; avoiding

dampness about the water fountains; avoiding puddles on the range, etc.

(3) Culling sickly birds as soon as noticed.

(4) Keeping the flock in good vigour by proper feeding and management. This is important because birds that are down in vigour are known to be more susceptible to coccidiosis than those that are vigorous and healthy.

(5) Using Nicarbazin, sulfaquinoxaline, Megasul or NFZ-Mix in the feed on premises (broiler plants) where coccidiosis constitutes a major disease problem.

Caution should be exercised in the use of both Nicarbazin and sulfaquinoxaline as coccidiostats. Nicarbazin should not be used in the ration of laying or breeding birds as it affects shell color and causes mottled yolks. Feeding sulfaquinoxaline at excessive levels reduces blood clotting time and appears, in some manner, to be associated with the occurrence of diffuse haemorrhages in various parts of the body (see Haemorrhagic Disease).

**Blackhead.**

Blackhead, also known as infectious enterohepatitis, is an acute or chronic infectious disease caused by the protozoan organism *Histomonas meleagridis*. The disease is of widespread occurrence, affecting principally turkeys, although chickens may also be affected. In turkeys of one to three months of age, the disease is rapid in its course and highly fatal. In adults the disease follows a chronic course, losses being but occasional.

The main symptoms of blackhead in young turkeys are: loss of appetite, increased thirst, poor growth, emaciation, weakness, drowsiness, ruffled feathers and drooped wings. Orange-brown diarrhoea may also be evident. The principal symptoms of this disease in adult birds are: progressive emaciation, sunken eyes, sluggishness and diarrhoea. Chickens affected with blackhead usually do not show any outward evidence of infection.

Autopsy findings in blackhead are confined to the liver and caeca (blind pouches of the intestine). The livers of affected birds are often enlarged and have round, sunken, ulcer-like areas of pink or yellowish color over their surfaces (see Fig. 36). The liver lesions frequently have a cart-wheel-like appearance. The caeca are usually very much enlarged and their walls are inflamed, thickened, and ulcerated (see Fig. 37). The presence of cheesy material in the caeca is also characteristic of this disease. While the symptoms and post-mortem findings described are definitely suggestive of blackhead infection, positive diagnosis is dependent upon microscopic examination of the liver and caeca of affected birds.

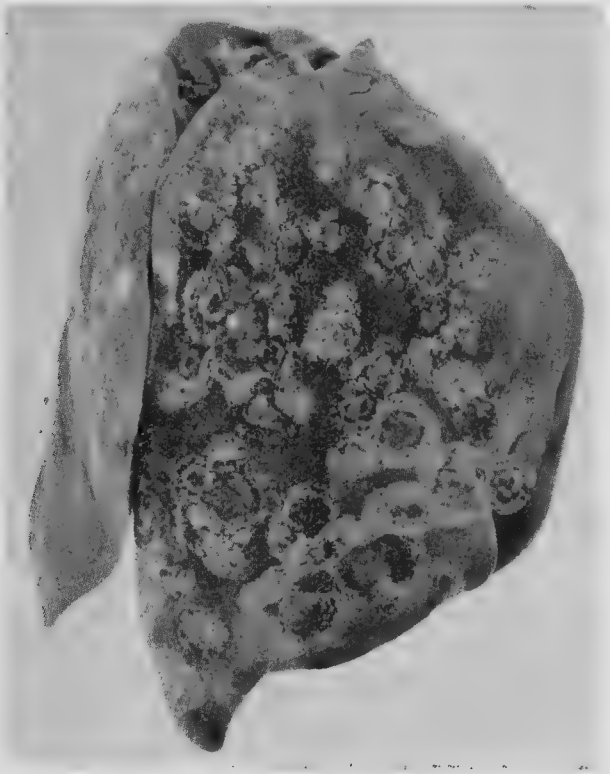


**Fig. 36— Liver of a turkey affected with blackhead. Note cart-wheel-like lesions.**
(After British Ministry of Agriculture and Fisheries.)

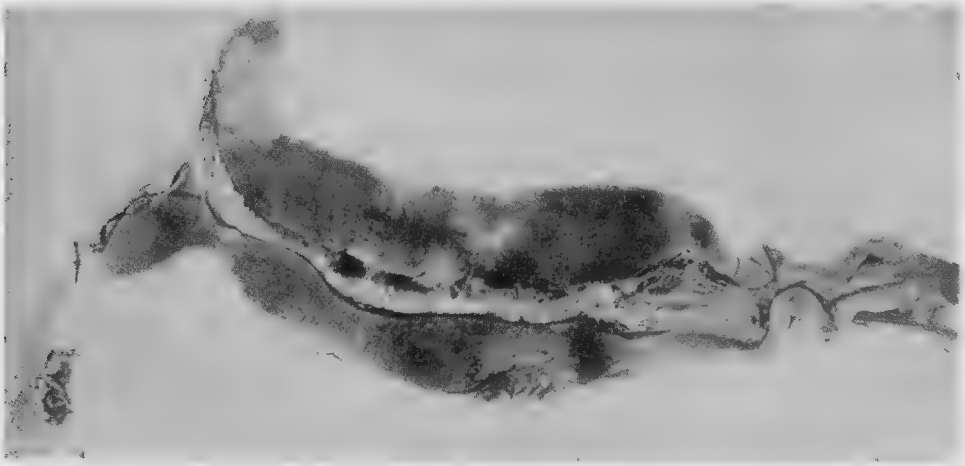


**Fig. 37—Caeca of turkey affected with blackhead. Note discolored diseased areas.**
(After Graybill.)

A few days after infection has taken place, *Histomonas meleagridis*, the causative organism, is passed in the droppings of affected birds, either in free state or enclosed in the egg of the caecal worm, *Heterakis gallinae*. In the free state the organism seldom survives more than twenty-four hours. However, when enclosed in the caecal worm egg, the organism is able to survive in the soil for more than one year. The principal agents responsible for the spread of this disease are infected or "carrier" chickens and turkeys.

Heaviest losses from blackhead are sustained in young turkeys. Losses in adult turkeys are but occasional.

At the first signs of an outbreak the entire flock should be fed a ration containing 0.05% of 2-acetylamino-5-nitrothiazole (6.6 lbs. of Enheptin-A Premix 15%, Lederle Laboratories Inc., per ton of feed) for a period of two weeks. By this time the outbreak should be under control, but the infection may still be present in the birds and on the premises. In order to prevent a further outbreak it is recommended that the preventive level of the drug (0.015% 2-acetylamino-5-nitrothiazole obtained by mixing 2 lbs. Enheptin-A Premix 15% per ton of ration) be fed and that the flock be transferred to clean ground, that is ground over which poultry have not ranged during the previous year.

Another drug that has been found to be highly effective in the control of blackhead is 1-ethyl-2-(5-nitro-2 thiazolyl) urea (Nithiazide, Merck and Co. Ltd.). For treatment purposes this compound is included in the ration at the 0.05% level.

The occurrence of blackhead may be prevented by rearing young turkeys on wire for the first ten to twelve weeks and then growing them on clean range, away from old turkeys and by keeping both young and old turkeys away from chickens or ground to which chickens have had access. Furthermore, since the blackhead organism enclosed in the egg of the caecal worm will remain alive for a much longer time than when not so protected, it should be obvious that preventive measures for blackhead should also include rigid control over caecal worm infestation on the farm (see Internal Parasites).

During recent years continuous inclusion of 2-acetylamino-5-nitrothiazole in the feed at the 0.015% concentration (2 lbs. Enheptin-A Premix 15% per ton of ration) or 1-ethyl-2-(5-nitro-2 thiazolyl) urea, (Nithiazide, Merck and Co. Ltd.) in the feed at the 0.0125% level has been recommended as a preventive measure for blackhead on farms where the disease is a serious problem. However, medication should not be considered as a substitute for the good management practices referred to above.

**Hexamitiasis.**

This is a disease to which poults of one to eight weeks of age are highly susceptible. It is caused by the protozoan parasite, *Hexamita meleagridis.*

The symptoms of this disease are not very specific. Affected poults appear listless and huddle together as if cold, chirping plaintively. The feathers are dry and ruffled. Foamy watery diarrhoea is frequently evident. Feed consumption is about normal but the birds lose weight as a result of improper digestion and assimilation of feed consumed. The peak of mortality is reached 7 to 10 days following the appearance of external symptoms. Internal examination reveals catarrhal inflammation of the duodenum (U-shaped part of the small intestine) and presence of foamy mucus in this portion of the intestine. Diagnosis should be based on the finding of *Hexamita meleagridis* in smears prepared from material taken from the upper portion of the intestine. Because the causative agent dies within a few hours after the poult dies it is necessary that live, sick birds be supplied to the laboratory for examination.

Many birds that recover from hexamitiasis become "carriers". These "carriers" look normal but constitute a menace to the well-being of subsequent groups of poults. In addition, as breeders, they may serve to perpetuate the infection on the farm by making possible the mechanical transfer of the infective agent to the next year's crop of poults. The number of Hexamita introduced from the "carrier" breeders to the first group of poults may be so small as not to produce outward evidence of disease in the poults. However, in the poult's intestine the causative agent multiplies rapidly. As a consequence, when the second group of poults arrive there will likely be sufficient Hexamita in the brooder house to produce the disease.

Aureomycin (Lederle Laboratories, Inc.) has been found to be fairly effective against the parasite that causes hexamitiasis. For treatment purposes the antibiotic should be administered in the feed for 14 days at the rate of not less than 200 grams of Aureomycin per ton of ration, while for preventive purposes the drug should be included in the feed on a continuous basis at the rate of 50 grams per ton of ration.

Two other materials have been found useful in the prevention of mortality from hexamitiasis. Furazolidone (Dr. Hess and Clark, Inc.) included in the feed at the rate of 100 grams per ton of ration for two weeks has been reported as a satisfactory treatment measure, while Tinostat (Dr. Salsbury's Laboratories) added to the feed, during the first twelve weeks of the poult's life, at the rate of

3 pounds per ton of feed has been found effective in preventing severe mortality on contaminated premises.

Good management and sanitation should be practised to minimize losses from hexamitiasis.

1. When possible, don't keep breeder turkeys and poults on the same premises. If breeder turkeys must be present isolate poults from them as completely as possible.
2. Avoid, if possible, multiple brooding. If the brooding facilities must be used for several hatches of poults, thoroughly clean and disinfect after each group is moved out of the brooder house.
3. Keep visitors out of the brooder houses. They may carry the causative parasite on their feet.

**Trichomoniasis.**

Trichomoniasis is a disease frequently encountered in turkeys. It is caused by protozoan parasites belonging to the genus Trichomonas.

External symptoms noted in this disease are: loss of appetite, haggard appearance, sagging wings, drooling from the mouth, depressed chest and yellowish foamy diarrhoea. Internal examination reveals inflammation of the gullet, crop and proventriculus (glandular stomach). Grayish or yellowish pyramid-like necrotic ulcers (see Fig. 38) are frequently noted in the gullet and crop.

The causative agent of this disease is passed in the droppings of infected birds. Birds that recover from the disease frequently become "carriers". Hence it is not advisable to use recovered birds as breeders as they may serve as a source of infection to the next year's crop of poults.

Since the causative agent is passed in the droppings of infected birds, sanitation is important in the control of an outbreak of this disease. Sick birds and apparently healthy birds should be segregated and both groups should be moved to clean, dry range. Copper sulphate should be added to the drinking water for two or three days and repeated after a few days if improvement has not been noted. The proper concentration of copper sulphate to use is 1:2000 (see Thrush for method of preparation of copper sulphate solution).

## FUNGOUS DISEASES

There are at least three diseases to which poultry are susceptible that are caused by fungi. These are: aspergillosis, favus and thrush.

**Aspergillosis.**

Aspergillosis, also known as brooder pneumonia, is a respiratory disease to which both young and adult birds are susceptible. The

Fig. 38—Trichomoniasis of upper digestive tract of turkey. (After Moynihan.)

disorder is caused by the fungus *Aspergillus fumigatus,* which is invariably present in mouldy feed and litter.

External symptoms of this disorder are: sluggishness, droopiness, loss of appetite, emaciation, sneezing, wheezing, and gasping for breath. In young birds the disease is very rapid in its course and usually fatal. In adult birds the disease is much slower to develop and less fatal. Post-mortem examination of afflicted birds reveals grayish, greenish or yellowish lesions in the trachea (windpipe), bronchi, lungs and air sacs. Positive diagnosis of this disease is dependent upon microscopic examination of affected tissue.

No specific treatment is known for birds affected with aspergillosis. In the event of an outbreak of this disease the feed and litter should be checked for the presence of mould. If the feed is at fault the troughs and hoppers should be cleaned and disinfected and refilled with fresh feed, free of mould. If, on the other hand, mouldy litter is the cause of the trouble the house should be thoroughly cleaned and disinfected and replenished with mould-free litter. Copper sulphate (bluestone) solution (0.5% copper sulphate in water) will be found an effective disinfectant for mould.

**Favus.**

Favus, or white comb, is a skin disease of young and adult birds, caused by the fungus *Achorion gallinae.* The disease is transmitted from one bird to another by direct contact.

Favus is characterized by the presence of dirty-white or yellowish scaly lesions on the comb, earlobes, face and wattles (see Fig. 39).

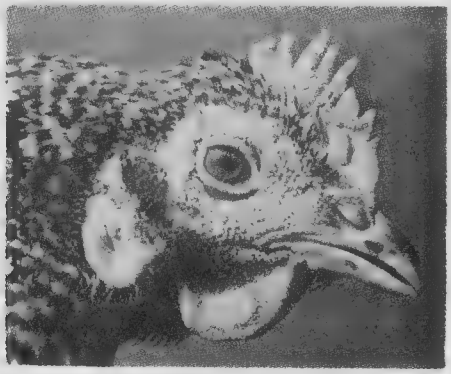


**Fig. 39—Favus. Note white scaly lesions on comb, face and wattles.** (After Bullis.)

In advanced cases the head and neck may be bare. Positive diagnosis of favus is dependent upon microscopic examination of affected tissue.

Birds afflicted with favus may be treated with formalized vaseline. Formalized vaseline is prepared as follows: melt a small amount of vaseline in a jar provided with a tight fitting cork, to the melted vaseline add 5% by weight of commercial formaldehyde, cork the jar tightly and shake until the vaseline has hardened. One or two applications of formalized vaseline are usually sufficient.

Another treatment that has been found effective consists of applying to the affected parts a mixture of one part tincture of iodine to six parts of glycerine. Two or three applications are usually necessary.

Favus is transmissible to man through skin abrasions. However, where a reasonable amount of care is exercised in handling affected birds there is little danger of contracting the disease.

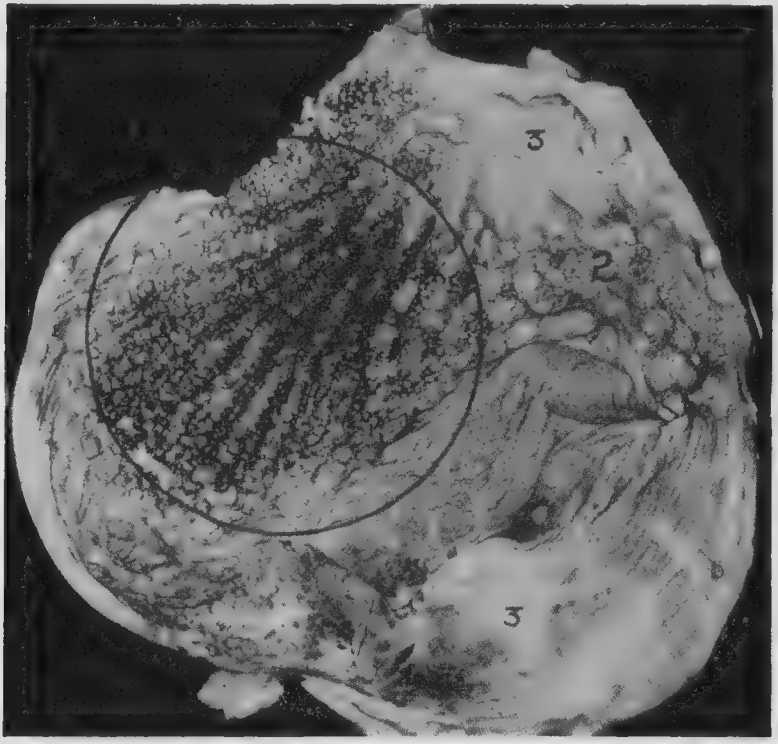
**Thrush.**

The term, thrush, is used to designate a fungous disease of fowls in which the mucous membranes of the digestive tract are involved. Several different species of fungi are capable of causing thrush.

One species, *Oidium pullorum,* attacks the mucous membranes of the mouth, pharynx, and upper larynx, causing cankerous growths. These cankerous growths differ from those encountered in diphtheritic fowl pox in that they can be removed without causing bleeding, whereas removal of pox cankers usually causes bleeding. In cases where the mouth and pharynx are involved, eating is made difficult, and as a result affected birds lose weight rapidly. On the other hand, where the upper larynx is involved, death by asphyxiation often results. Treatment of this form of thrush is readily accomplished by removing all cankerous growths and painting the areas from which the cankers were removed with tincture of iodine.

Two other species of fungi, *Monilia albicans* and *Monilia krusei,* attack the gullet, crop (see Fig. 40), proventriculus, gizzard, intestine, and anus, causing severe ulceration. Chicks and poults affected with this form of thrush die rapidly. Adults, on the other hand, are much slower to die. Treatment of advanced cases of this form of thrush is useless. Treatment of less advanced cases may be accomplished by giving copper sulphate (bluestone) in the drinking water. The proper dilution of copper sulphate for this purpose is 1:2000. Dilutions stronger than 1:500 should not be given as they are toxic. The 1:2000 copper sulphate solution should be kept before the birds as their only drink until the trouble has cleared up. The solution should be supplied in wooden troughs or earthenware, not in metal ware, as copper sulphate corrodes metal. During the treatment the houses and equipment should be kept scrupulously clean.

A convenient method of preparing a 1: 2000 dilution of copper sulphate solution is as follows: Make a stock solution by dissolving one pound of copper sulphate in one gallon of soft water (rain water). If soft water is not available, add one teaspoonful of concentrated hydrochloric acid or one cup of vinegar to the water before




Fif. 40—Inner surface of crop of fowl affected with thrush. The area enclosed by circle is severely affected while the area marked 2 shows more severe changes from the disease. The areas marked 3 show healthy tissue. (After Durant.)

adding the copper sulphate. Bring the copper sulphate into solution by heating. Store this stock solution in glass or crockery ware. To make a 1: 2000 dilution add one tablespoonful of the stock solution to one gallon of soft water. If hard water is used enough hydrochloric acid or vinegar should be added to the water to prevent precipitation of the copper. The amount of acid required will vary with the hardness of the water. Not more than one teaspoonful of hydrochloric acid should be added to each gallon of water.

Positive diagnosis of either of these two forms of thrush can only be made by microscopic examination of affected tissue.

The fungi responsible for both forms of thrush are passed in the droppings of affected birds. Susceptible birds coming in direct contact with infected droppings contract the disease.

Trouble from thrush may be avoided by seeing that feed and litter used for poultry are free of mould and by keeping houses and equipment in sanitary condition.

## NUTRITIONAL DISORDERS

Nutritional disorders arise as a result of a deficiency or an excess of one or more nutrients in the diet. Among the nutritional disorders that are commonly encountered in poultry are: nutritional roup, riboflavin deficiency, rickets, perosis, gout, obesity and haemorrhagic disease.

### Nutritional Roup.

Unlike ordinary roup (coryza), nutritional roup is not infectious but is caused by a lack of vitamin A in the diet.

Nutritional roup is most often observed under conditions where rations inadequate in vitamin A are fed for long periods of time to birds kept in confinement. Usually the disorder appears when birds are fed excessive amounts of grain. This happens when only grain is fed or when the concentrate is diluted with grain to such an extent that insufficient vitamin A is provided. This disorder is seldom encountered in birds on good range. This, of course, is because good range is an excellent source of vitamin A activity.

The symptoms of vitamin A deficiency in young chicks are: loss of appetite, poor growth, staggering gait, soreness of the eyes, diarrhoea, and drowsiness. The manifestations of nutritional roup in young turkeys are the same as those listed for chicks except that young turkeys may show one additional symptom, namely, swelling of the sinuses. Deficiency of vitamin A in the diet of laying birds causes low hatchability of eggs, decreased egg production and eventually, cessation of egg production. The symptoms of prolonged vitamin A deficiency in adult birds are: loss of appetite, incoordinated gait, soreness and wateryness about the eyes and accumulation of cheesy material in the eyes (see Fig. 41), swollen sinuses, impaired vision, droopiness, and sleepiness.

Post-mortem examination of afflicted chicks reveals white deposits (urates) in the kidneys and cloaca (chamber just inside vent) and similar deposits on the heart, liver and other visceral organs. Autopsy findings in affected birds are not constant. An internal examination of adult birds suffering from nutritional roup shows urate deposits in the kidneys (see Fig. 42) and cloaca, and similar deposits on the heart, liver and other visceral organs and the pres-

**Fig. 41—Cheesy material in the eye of a fowl affected with nutritional roup.** (After Beach.)

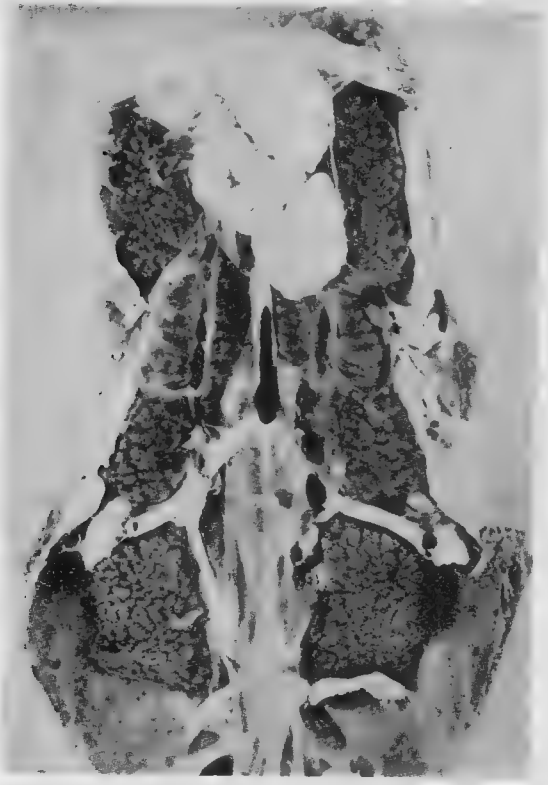


**Fig. 42—Urate deposits in kidneys of fowl afflicted with nutritional roup.** (After Beach.)

ence of white pustules in the mouth, pharynx and oesophagus (gullet) (see Fig. 43). While diagnosis of advanced stages of nutritional roup can readily be made on the aforementioned symptoms and post-mortem findings, diagnosis of less advanced stages is impossible on that basis. To diagnose less advanced stages properly one must have knowledge of the composition of the

**Fig. 43—White pustules in mouth, pharnyx and oesophagus of bird affected with nutritional roup.** (After Beach.)

ration that is being fed and of the range conditions under which the birds are kept.

Losses from nutritional roup in Alberta are relatively serious. The death loss from advanced nutritional roup is relatively insignificant when compared to the loss sustained through poor growth

and development of young stock, poor egg production of laying stock, poor hatchability of eggs from breeding stock and low resistance to disease in all classes of poultry.

Trouble from nutritional roup may be avoided by including an adequate amount of vitamin A in the diet. The requirements for vitamin A for various classes of chickens and turkeys are as follows: starting and growing chicks 1,200 I.U. (International Units), laying and breeding hens 2,000 I.U., starting and growing poults 2,400 I.U., and breeding turkeys 2,400 I.U. per pound of feed.

In formulating rations, levels considerably higher than the above are usually included. The excess over the requirement is added in order to compensate for loss of the vitamin in storage, variation in composition of feeds, and individual differences in requirement.

The principal sources of vitamin A activity in poultry rations are alfalfa meal, cereal grass, fish oil, and synthetic vitamin A. In order to supply the requirements of poultry, 2 to 5 percent good quality alfalfa meal or cereal grass is included in starting and growing rations and from 5 to 7½ percent in laying and breeding mashes. These levels are used along with appropriate amounts of fish oil and/or synthetic vitamin A to supply the requirements for vitamin A.

In addition to obtaining vitamin A activity from alfalfa meal, cereal grass and fish oil, poultry obtain vitamin A activity from green feed, yellow carrots, and yellow corn. When an abundance of these vitamin A carrying feeds is available for feeding, the alfalfa meal and/or cereal grass in the mash may be reduced materially.

Treatment of nutritional roup may be accomplished by supplying a high level of vitamin A in the diet of the affected birds. The vitamin A content of the mash should be well in excess of the requirements, and in addition, if available, the affected birds should be given all the fresh green feed that they can consume.

**Riboflavin Deficiency.**

Curled-toe paralysis is a disorder encountered in chicks receiving inadequate amounts of riboflavin in their diet. The condition is characterized by paralysis of the legs and inward curling of the toes (see Fig. 44).

Besides being necessary for the prevention of curled-toe paralysis, riboflavin is essential for the promotion of normal growth, egg production and hatchability.

Feedstuffs which contain substantial amounts of this vitamin include buttermilk powder, dried whey, skimmilk powder, alfalfa meal, cereal grass and certain by-products of the fermentation industry.

**Fig. 44—The chick on the left received an adequate amount of riboflavin in its diet while the one on the right did not. Note curled toes of chick on right. Both chicks were four-weeks-old when photographed.** (After Clandinin and Robblee.)

The riboflavin requirements per pound of feed of various classes of poultry are as follows: starting chicks 1.3 mg., growing chicks 0.8 mg., laying hens 1.0 mg., breeding hens 1.7 mg., starting poults 1.7 mg., and breeding turkeys 1.5 mg.

The above requirements are usually met by adding pure riboflavin or a riboflavin concentrate to the ration. The amount of riboflavin added will depend on the ingredients in the ration and the purpose for which the ration is intended. The addition of riboflavin at the rate of 2 grams per ton of starter, 1 gram per ton of growing or laying mash or 3 grams per ton of breeding mash will usually meet the requirement for riboflavin.

Chicks afflicted with curled-toe paralysis may be brought back to normal by placing them on a diet adequate in riboflavin and by greatly increasing their riboflavin intake for a period of about a week. The latter may be accomplished by adding extra riboflavin to the mash or drinking water.

**Rickets.**

Rickets is a disorder characterized by faulty mineralization. It may be caused by (1) vitamin D deficiency, (2) calcium deficiency, (3) phosphorus deficiency, or by (4) improper balance between calcium and phorphorus in the ration.

The principal symptoms of rickets in growing birds are: staggering gait, reluctance to stand or move, enlarged joints, bent legs,

soft beaks, crooked breastbones, poor growth and general unthrift. Post-mortem examination reveals a beaded rib condition and piling up of cartilage at the ends of the long bones. Bone ash analysis shows low mineral content of bones. In layers, rickets results in impaired hatchability and egg production, and in the production of thin-shelled abnormally small eggs. Rarification of bones also takes place with the result that breastbones as well as other bones become crooked.

Rickets may be avoided by providing a ration adequate with respect to vitamin D, calcium and phosphorus content. The vitamin D requirements per pound of feed of various classes of chickens and turkeys are as follows: starting and growing chicks 90 I.C.U. (International Chick Units), laying and breeding hens 225 I.C.U., starting and growing poults 400 I.C.U., and breeding turkeys 400 I.C.U. The calcium and phosphorus requirements of starting and growing chicks are 1.0% calcium, 0.6% phosphorus; of laying and breeding hens are 2.25% calcium, 0.6% phosphorus; of starting poults are 2.0% calcium, 1.0% phosphorus; of growing turkeys are 2.0% calcium, 1.0% phosphorus, and of breeding turkeys are 2.25% calcium, 0.75% phosphorus.

The vitamin D requirements (given above) may be met by either permitting birds to have access to direct sunlight (the ultra-violet rays of the sun act on certain substances present in the skin to produce vitamin D) or by including a standardized fish oil or a vitamin $D_3$ concentrate in the mash at the proper level. For example, the addition of 0.1% of a fish oil containing 300 I.C.U. of vitamin D per gram to a chick starter or 0.5% to a chicken laying or breeding mash will satisfy the requirements for vitamin D. The calcium and phosphorus requirements of growing stock may be met by including 1% limestone in the average starter, and 1% limestone and 1% bonemeal in the average growing mash. Oyster shell and bonemeal should be fed free choice after the feeding of scratch grain has been commenced. The calcium and phosphorus requirements of laying and breeding stock may be satisfied by including 2% limestone and 2% bonemeal in the average laying and breeding mash and by feeding oyster shell free choice. When protein supplements of vegetable origin are used in compounding poultry mashes, extra bonemeal should be included in the mash. This is necessary in order to compensate for the low mineral content of protein supplements of vegetable origin.

Where trouble from rickets is encountered, the mineral balance of the ration should be checked, and if necessary, corrected. Then the vitamin D content of the mash should be increased to approximately twice the level required. Following correction of the dis-

order, the vitamin D content of the mash should be reduced to the proper level.

**Perosis.**

Perosis, also known as slipped tendon, is a disorder that is frequently noted in growing birds, particularly those raised in confinement. It is prone to occur on rations that contain excessive amounts of calcium or phosphorus or inadequate amounts of manganese. A deficiency in the ration of a number of B-vitamins including choline niacin, folic acid, pantothenic acid or biotin may also result in an increased incidence of perosis.

The condition is characterized by bowing or twisting of the legs, slipping of the Achilles' tendon ( large tendon of the hock joint) from its normal position (see Fig. 45) and enlargement and flattening of the hock joint.

**Fig. 45—Chick affected with perosis.** (After Clandinin and Robblee.)

There is no method of treating birds affected with perosis. Control of losses from this disorder must therefore be effected through prevention. Perosis may be avoided by including proper amounts of calcium and phosphorus (see Rickets), and manganese (0.005%), and necessary B-vitamins in the ration.

How to obtain required levels of calcium and phosphorus is explained under Rickets, paragraph 4. To meet the manganese requirement (given above), 1/4 of a pound of manganese sulphate should be added to each ton of ration. The requirements for choline, niacin, folic acid, pantothenic acid and biotin are usually met in

starting rations for replacement stock without special supplementation; however, in specialized broiler rations and in turkey starters it is advisable to add supplementary amounts of some or all of these vitamins.

**Gout.**

Gout is a chronic ailment that may affect either young or adult birds. It is believed to be caused by a disturbance in protein metabolism. Gout is most frequently encountered in birds on high protein rations. Close confinement, with accompanying lack of exercise, renders birds more susceptible to this disorder.

Two types of gout are recognized, visceral and articular. The visceral form is characterized by bluish comb, sluggishness, anemia, emaciation and diarrhoea. Internal examination of birds affected with visceral gout reveals kidneys which are often enlarged, pale in colour and coated with whitish material (uric acid salts). The heart, liver and other visceral organs may also show similar deposits. Articular gout, on the other hand, is characterized by lameness and swelling of the leg, feet and wing points. The swellings frequently break outward, discharging white or grayish yellow masses of uric acid salts. Diagnosis of gout is dependent upon microscopic examination of the whitish deposits in acidified water.

Trouble from gout may be prevented by avoiding excess protein feeding and by providing ample opportunity for exercise. The approximate protein requirements of various classes of chickens and turkeys are: growing chickens—20% to 6 weeks, then level gradually decreased to 14 to 15% at sexual maturity; growing turkeys—28% to 6 weeks, then level gradually decreased to 16% by 20th week; fattening chickens or turkeys—14%; laying chickens or turkeys—15%.

Treatment of birds affected with gout may be effected by reducing the protein in the diet, supplying an abundance of green feed and by providing ample opportunity for exercise.

**Obesity.**

Obesity is a term used in referring to birds that carry an excess of abdominal fat, and is most frequently encountered in layers and breeders of meaty type. The condition is generally attributed to excessive carbohydrate (grain) feeding. The feeding of large proportions of wheat and barley also seems to cause the condition.

Trouble from obesity may be avoided by feeding rations which do not contain too high a level of productive energy in relation to the protein level in the ration. This can be achieved by feeding rations containing moderate levels of wheat and barley and recommended levels of laying or breeding supplement.

**Haemorrhagic Disease.**

A condition termed "haemorrhagic disease" or "haemorrhagic syndrome" has been noted frequently in commercial broiler flocks during the past few years. In some instances losses have been heavy.

While the exact cause of the disease is not known, a number of factors have been implicated. The appearance of the condition has coincided with the use of high energy rations for broilers. Since these rations typically contain less alfalfa meal than lower energy rations, and since alfalfa meal is a good source of vitamin K (a factor required for the normal clotting of blood), it was thought, at first, that the condition might be caused by an uncomplicated vitamin K deficiency. Laboratory studies indicated that the condition could be prevented by supplementing rations with viatmin K; however, in the field many outbreaks failed to respond to vitamin K therapy. It now appears that several other factors may influence the appearance of the disease. It is known that feeding of sulfa drugs increases blood-clotting time, and that to counteract this effect increased levels of vitamin K are required. It has been suggested that over-medication with antibiotics alone or in combination with sulfa drugs may result in a deficiency of vitamin K. It appears too, that the occurrence of haemorrhages may be influenced and complicated by such factors as: changes in the processing procedures used for various ration ingredients, specific infections, environmental stress condition, and possibly by toxins produced by moulds.

The symptoms of the disease are not specific. Haemorrhages are found under the skin or in the organs of the bird. The lesions are noted most commonly in the breast (see Fig. 46) and thigh regions, but may also occur in the covering of the brain (see Fig. 47), the vicinity of the eyes (see Fig. 48), the respiratory organs and along the digestive tract. The extent of the haemorrhages varies greatly; in some cases the lesions are small and no mortality results, in others the haemorrhages are extensive and mortality may be high. An increase in blood-clotting time (a symptom occurring in a vitamin K deficiency) is encountered in some cases but not in others. The appearance of the disease is sometimes accompanied by an anemia and by changes in the color of the bone marrow.

No clear-cut method of prevention can be outlined at the present time. Control measures adopted should recognize that a number of factors may be responsible for the appearance of the disease. In order to counteract the possibility of a vitamin K deficiency the level of alfalfa meal or cereal grass in the ration should be increased. Addition of synthetic vitamin K may also be bene-

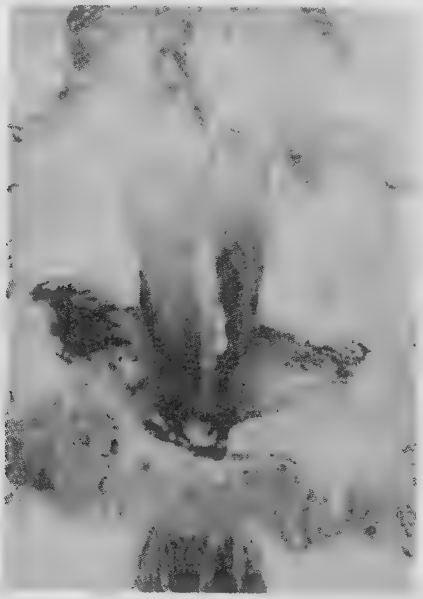


**Fig. 46—Haemorrhages in the breast and thigh muscles as noted in haemorrhagic disease.** (After West Virginia University Agricultural Experiment Station.)

**Fig. 47—Haemorrhagic disease. Haemorrhages in the membrane covering the brain as seen after removal of a portion of the skull.** (After West Virginia University Agricultural Experiment Station.)

ficial. As further precautions against an outbreak of "haemorrhagic disease", the birds should be fed a well-balanced ration supplemented with adequate levels of vitamins; efforts should be made to avoid environmental stresses such as extremes of temperature; and care should be taken to avoid over-medication with antibiotics and sulfa drugs. In this latter regard the disease has often been confused with coccidiosis because of the appearance of blood in the droppings. Treatment for coccidiosis under such circumstances has sometimes led to an increased rate of mortality.

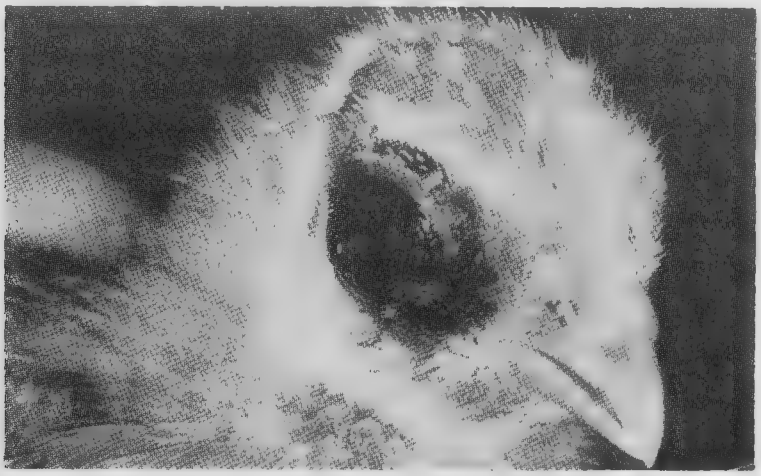


**Fig. 48—Haemorrhages in the vicinity of the eye in a chick affected with haemorrhagic disease.** (After West Virginia University Agricultural Experiment Station.)

## MISCELLANEOUS DISORDERS

There are a number of disorders and vices which are not infrequently encountered in poultry and which therefore warrant brief consideration. They include: beak canker, beak necrosis, impaction of the crop, pendulous crop, impaction of the gizzard, bumblefoot, ascites, reproductive disorders, and various forms of cannibalism.

### Beak Canker.

Beak canker is a condition that is occasionally noted in adult birds. The disorder arises as the result of mechanical injury followed by secondary infection. The most common seat of this trouble is the beak angle. Quite a large swelling may be present. Treatment of beak canker is comparatively simple since it merely entails removing the scab, pressing out the core and pus, swabbing the hole with tincture of iodine and feeding the treated bird(s) whole grain for a week or two.

**Beak Necrosis.**

This is a disorder that is encountered in chicks fed mash that has been ground too finely. The finely ground feed accumulates in and around the mouth causing inflammation of the tissues and predisposing to secondary infection. As the condition progresses the beak becomes deformed (see Fig. 49). Trouble from beak necrosis may be avoided by feeding mashes of granular texture.

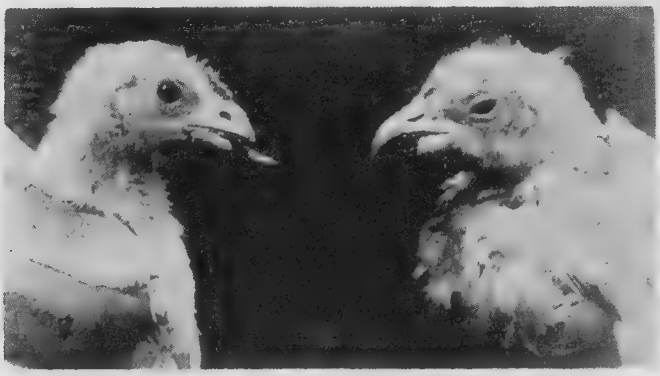


**Fig. 49—Chicks affected with beak necrosis. (After W. A. Maw.)**

**Impaction of the Crop.**

This condition, commonly known as crop-bound, is caused by obstruction resulting from the eating of feathers, straw, long or dead grass or other indigestible material. The symptoms include: loss of appetite, inactivity and general distress. The crop is distended and feels doughy. A sour smell is usually present.

In some cases crop impaction can be corrected by giving an exclusive diet of sour milk, in other cases the condition can be relieved by administering vegetable oil, suspending the bird head down and working the material in the crop out through the mouth. However, in most cases, a minor operation is necessary in order to alleviate the condition. The operation referred to consists of making an incision, about one inch in length, in the upper front surface of the crop (both the outer skin and crop wall are cut), removing the impacted material through the opening, washing out the crop with warm water to which antiseptic has been added and sewing up the crop wall and outer skin separately with three or four stitches (each stitch is tied separately.) Catgut, silk or cotton thread may be usel for sewing. It is not necessary to remove the stitches. For two or three days following the operation only soft feed should be fed.

Trouble from crop impaction can be avoided to a considerable extent by keeping mash free from straw and by observing reasonable care in turning birds out in the spring. In the latter connection it is not advisable to turn birds out before grass growth is fairly well under way. Turning birds out before growth has started often results in crop impaction caused by birds consuming large quantities of dead grass.

**Pendulous Crop.**

Pendulous crop is a condition encountered in both chickens and turkeys although more frequently in the latter. In this disorder the crop is very much enlarged and hangs down in front of the affected birds (see Fig. 50).

**Fig. 50—Pendulous crop in a turkey.** (After General Mills.)

Pendulous crop is an inherited abnormality, which is more prone to occur under certain conditions. The conditions that predispose to pendulous crop are: over-consumption of water, milk or bulky feed, high temperature and low humidity.

Trouble from pendulous crop can be minimized by using as breeders only birds not affected with the abnormality and by avoiding excessive water, milk or bulky feed consumption. Various methods of treating birds affected with pendulous crop have been tried, but none can be highly recommended. Supporting the crop

with a cloth or canvass will give temporary relief. However, in most instances it is best to kill and market affected birds as soon as they are detected in the flock.

**Impaction of the Gizzard.**

This is a disorder quite frequently noted in birds getting an excessive amount of oat and barley hulls in their feed, or in birds getting a moderate amount of oat and barley hulls in their feed and little or no grit, or in birds that are low in vigour as a result of disease or parasites. Lack of appetite, reluctance to move about and general unthrift are the main symptoms of this disorder. Post-mortem examination of affected birds reveals the presence of excess fibrous material in the gizzard.

Treatment of affected birds may be successful in some instances, and not in others. Recovery is dependent upon the severity of the impaction and the vigour of the individual. Where impaction has resulted from feeding a ration containing too much oat and barley hulls or too little grit, treatment consists of feeding a ration of lower oat and barley hull content and supplying grit. On the other hand, when impaction is attributable to some other cause, treatment consists of correcting the primary cause.

**Bumblefoot.**

The term, bumblefoot, is used in referring to a swelling of the ball of the foot. The condition may arise as a result of foot injury (such as that sustained by birds jumping down from high roosts or nests on to bare floors) and subsequent infection by non-specific infective agents, or may arise as the result of a gouty condition.

Before attempting to treat birds for bumblefoot the predisposing cause or causes must be determined and eliminated. Then the affected birds should be examined and segregated into two groups: those showing the presence of a core, and those not. The birds in the former group should be treated, while those in the latter should be placed back in the flock and examined periodically to see whether recovery or core formation has taken place. Treatment consists of removing the scab, pressing out the core and pus, swabbing the hole with tincture of iodine, applying sulfa ointment and bandaging. The treatment should be repeated if deemed necessary. In severe cases poulticing may be necessary to bring relief.

**Ascites.**

Ascites, also known as dropsy, is a condition often found accompanying enteritis (inflammation of the intestine), peritonitis (inflammation of membrane covering internal organs), fowl tuberculosis, and other diseases affecting the visceral organs. The disorder is not caused by a specific agent.

Ascites is characterized by accumulation of serous fluid in the body cavity and its presence or absence is readily ascertained by palpating the abdomen.

Treatment of birds affected with ascites is useless. When this trouble is detected, a post-mortem should be performed to determine the primary cause in order that appropriate control measures may be adopted.

**Reproductive Disorders.**

The principal reproductive disorders encountered in poultry are: ruptured yolk, intra-ovulation, impacted oviduct, ruptured oviduct, internal laying, egg bound, prolapsus and cloacitis.

*Ruptured Yolk.*—This is a condition occasionally encountered in pullets that have been laying at a high rate. Outward evidences of this disorder are sagging of the abdomen and discoloration of the comb. Post-mortem examination shows large quantities of yolk material free in the abdomen. In some cases *Pasteurella multocida*, the fowl cholera organism, has been singled out as the cause, whereas in other instances, excessive production and high roosts have been considered responsible for the trouble.

*Intra-ovulation.*—Intra-ovulation refers to the ovulation (laying) of unbroken yolks in the body cavity. The abnormality is usually caused by disfunction of the oviduct (egg tube), although it may also be caused by high roosts or by forcing birds to lay beyond their productive capacities. Birds affected with this disorder look like layers and regularly sit on the nest but do not always lay eggs.

*Impacted Oviduct.*—Impacted oviduct is another reproductive disorder that is quite commonly encountered. This condition is caused by the disfunction of the oviduct and is characterized by the presence of hardened yolk and albumen (egg white) in the oviduct.

*Ruptured Oviduct.*—This is a disorder that often develops subsequent to impaction of the oviduct. The break in the wall of the oviduct usually occurs just above the impaction. Birds afflicted with this condition generally lay their eggs into their body cavities.

*Internal Laying.*—Internal laying refers to the laying of complete eggs in the body cavity. The condition may arise as the result of impaction of the oviduct, rupture of the oviduct or disfunction of the oviduct. The abdomen of an internal layer is usually quite heavy and causes the bird to assume a duck-like posture. Upon palpation, whole or broken eggs may be felt in the abdomen. Internal examination confirms their presence.

Treatment of birds affected with ruptured yolk, intra-ovulation, impacted oviduct, ruptured oviduct or internal laying is in most instances useless. It is much better to attack these troubles from

the preventive angle. Keeping birds in good health by proper feeding and management and avoiding excessive production (over 80%) and high roosts (over three feet off the floor) will do much to prevent losses from these disorders.

*Egg Bound.*—This is a term used in referring to a bird that is unable to expel her egg. The disorder may be the result of temporary paralysis of the expelling muscles or to an attempt on the part of the bird to lay too large an egg. The condition can usually be remedied by removing the affected bird to warm quarters, and applying vaseline or vegetable oil to tissues around the vent. In instances where this procedure proves unsuccessful, puncturing the egg through the vent with a blunt tool and removing the shell is recommended.

*Prolapsus.*—Prolapsus, also called blow-out, is a disorder to which very early maturing pullets and pullets just coming into production seem highly susceptible. Birds carrying an excessive amount of abdominal fat and laying at too high a rate, and birds that have been egg bound also seem to be susceptible to this disorder. Prolapsus is characterized by eversion of the cloaca (chamber just inside the vent) and lower oviduct. Birds that have prolapsed, if discovered immediately, can usually be saved by carefully washing and greasing the protruding tissue and pushing it back into place. The oviduct will go back into place more quickly if the affected bird is suspended by the feet for a short time. Affected individuals should be kept separate from the rest of the flock for a week or so. Birds that have prolapsed and that are not discovered soon after, are usually disembowelled and killed by their pen mates. Trouble from prolapsus may be minimized by avoiding too early maturity in pullets, excessive abdominal fat in pullets and hens, and excesses of production (over 80%).

*Cloacitis.*—Cloacitis, or vent gleet, is generally considered to be a highly infectious venereal disease to which both chickens and turkeys are susceptible. The cause of cloacitis has not as yet been determined. The condition is characterized by swelling, inflammation and ulceration of the cloaca and vent, and by an anal discharge with an obnoxious odour. The disease is believed to be spread from one female to another by male birds.

Cloacitis is very resistant to treatment. If the trouble is detected early in the course of its development, recovery may be effected by applying a suitable disinfectant or sulfa ointment repeatedly to the vent. On the other hand, if the disorder is not noticed early in the course of its development, recovery by treatment is uncertain. Among disinfectants that are recommended for treatment of cloacitis are: 3% chromic acid, 3% silver nitrate, 5% carbolic acid, 1:1000

corrosive sublimate, 15% argyrol and 2% carbolic ointment. As a general rule it is advisable to dispose of affected birds as soon as detected.

**Cannibalism.**

Chickens are decidedly cannibalistic. It seems that once a chicken gets a taste of blood or raw flesh, it must have more of it even at the expense of one or more of its pen mates.

*Toe Picking.*—Baby chicks are subject to a form of cannibalism known as toe picking. This vice may be brought on by not having feed down for chicks on arrival at the brooder house, by letting feeding troughs be empty for too long a period, by idleness, by overcrowding, by overheating, by having too much light in the brooder house, or by permitting sun spots in the brooder house. Where toe picking has broken out, the predisposing cause should be determined and corrected. Next the windows should be covered with red tissue paper to exclude all white light. The red light prevents, to a considerable extent, the chicks from seeing the blood on the toes of injured chicks, and so the habit is curbed.

*Tail Picking.*—This is another type of cannibalism that is encountered in growing chickens. It often follows toe picking. It may also occur in older chickens. Among the factors that predispose to tail picking are: idleness, overcrowding, overheating, too close confinement, insufficient feeder space and letting feed troughs be empty for too long a period. Any factor that causes the chicks to become restless may be considered as predisposing to tail picking. Where tail picking has broken out the predisposing cause should be determined and corrected. Next, all the tail-picked birds, the "pickers" and a number of chicks selected at random should be smeared on the tail and wings with some anti-picking material such as pine tar or a mixture of 4 ounces of vaseline plus ¼ ounce of carmine plus ½ ounce of aloes. If the chicks are not running outside and weather conditions are such that they might be, every effort should be made to get them outside. Giving the chicks free range, and avoiding overcrowding will do much towards bringing this vice under control.

Two further aids to bringing an outbreak of tail picking under control are the hanging of red buttons from the ceiling at a height such that the chicks will have to jump to pick at them, and the feeding of salt in the drinking water. In the latter connection it has been found that one tablespoon of salt should be added to each gallon of drinking water and that the birds being treated should have access to the salt water on two or three forenoons, each three days apart.

*Feather Picking.*—A further cannibalistic habit is feather picking. This habit may be encountered in growing chickens, laying

chickens or in turkeys. It is most prone to occur where large numbers of birds are kept closely confined.

When feather picking is present in a flock, birds may be seen to go about picking out feathers from the backs, tails or from around the vents of other birds. In laying flocks, if the vice has not been recognized at an early stage, the backs and abdomens of most of the birds in the flock may be completely devoid of feathers (see Fig. 51), while in the case of turkeys the backs may be bare and

**Fig. 51—Note nude condition of back region typical of feather picking.** (After Richter.)

the tails may be literally eaten off. Further, many of the turkeys that have been picked on the back show evidence of dark pigmentation in the skin over the back which causes them to be graded down at market time.

Feather picking is believed to be brought on by overcrowding, close confinement, insufficient feeder space or by lack of one or

more nutrients in the diet. Absence of the disorder from flocks has been associated with the feeding of liberal amounts of oats (30%) in the ration. This beneficial effect may be the result of a better energy: methionine balance in the ration rather than to some specific nutrient in oats.

**Fig. 52—Debeaked turkey. Upper mandible cut back to nearly half-way between tip of beak and nostril.** (After West Virginia University Agricultural Experiment Station.)

Outbreaks of feather picking may be prevented or brought under control by avoiding overcrowding, providing adequate feeding space and including oats in the diet at the proper level or supplementing the ration with .05 to .1% DL-methionine and by debeaking (see Fig. 52) or using anti-picking devices (obtainable from the larger poultry supply houses) on the beaks of the birds (see Fig. 53 and 54) to prevent them from picking. Where debeaking is used as a preventive or curative measure the upper beak should be removed to a point about half-way between the tip of the beak and the nostrils (see Fig. 52) by means of an electric debeaker (see Fig. 55) procurable from the larger poultry supply firms.

*Vent Picking.*—Another vice which chickens frequently acquire is vent picking. Vent picking often breaks out following the disembowelling of two or three birds that have prolapsed. It may also accompany feather picking or it may have its origin with birds picking others that are laying. Vent picking is a vice that must be given prompt attention, otherwise serious losses may be sustained.

**Fig. 53—Anti-picking devices ("Pikgard") attached through the nostrils. Other patented anti-picking devices ("Hen Specs", "Helmets", "Anti Pix" and "Stop Picks") are available. (After Brunett.)**

A few offenders in a flock can teach the vice to the entire flock in a very short time, and in the course of a few hours many birds will have been disembowelled, and practically every bird in the flock will be bleeding at the vent. The vice may be brought under control

by debeaking ( see Fig. 52 and 55) all of the birds in the flock or by placing anti-picking devices (obtainable from the larger poultry supply houses) on the beaks of the birds (see Fig. 53) to prevent them from picking. Electric debeakers (see Fig. 55) may be obtained from most poultry equipment supply houses. Birds that have already been picked should have their vents smeared with an anti-picking material such as one of the materials referred to under Tail Picking. It has also been shown that adding extra synthetic methionine, an amino acid present in relatively large amount in fish meal, to the laying ration is helpful in bringing an outbreak of vent picking under control. The proper level of supplementary DL-methionine to use is .05 to .1 percent. Where synthetic methionine is not readily available supplementing the diet with 3 to 5% fish meal and making a corresponding reduction in the amount of meat

**Fig. 54—Anti-picking device "Turkey Bit" inserted under the upper mandible and through the nostrils with special pliers.** (After West Virginia University Agricultural Experiment Station.)

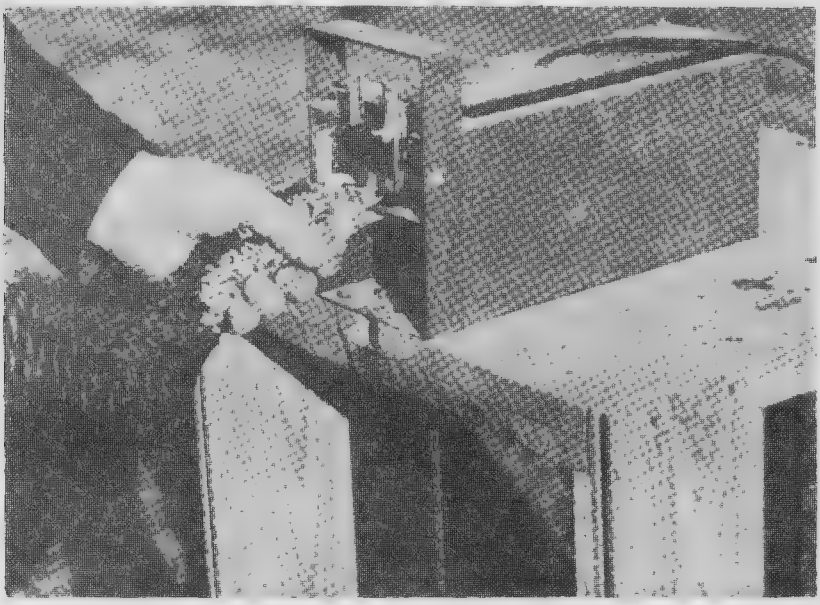


**Fig. 55—Electric debeaker. The up and down movement of the hot cutting and searing bar is controlled by a foot pedal.** (After West Virginia University Agricultural Experiment Station.)

meal in the ration might help to correct the condition. Darkening the nests or using the "Community-Type" nest and giving salt in the drinking water (see Tail Picking) will also aid in bringing this habit under control.

## EXTERNAL PARASITES

External parasites affect poultry adversely. The irritation caused by their movement, gnawing, and sucking, leads to restlessness in the bird. Restlessness in turn tends to throw the bird out of physiological balance. When this occurs, digestion and assimilation are slowed up. As a result, feed consumption drops. In young birds the decrease in feed consumption is paralleled by a marked decrease in the growth rate. Severe infestation may even end in death. In adult birds the decrease in feed consumption is coupled with a decrease in body weight and a falling off in egg production. In general, external parasites tend to bring about a state of unthriftiness, lowered vitality and lowered resistance to disease. There are two main types of external parasites that affect poultry, namely, lice and mites.

### Lice.

Lice are small yellowish or grayish flattened insects (see Fig. 56). Their bodies are divided into three regions: head, thorax and

abdomen. They have three pairs of legs and their mouths are equipped for biting and cutting. They do not suck blood, but feed on bits of feather, scales from the skin, and dried blood.

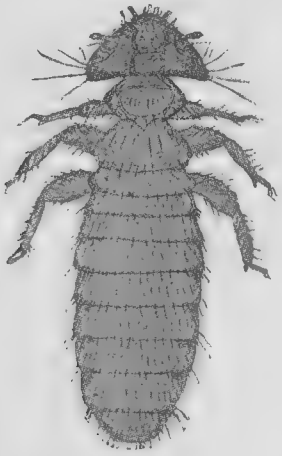


**Fig. 56—Common body louse of chickens.** (After Osborne.)

There are about fifty species of lice that infest domestic poultry. The common body louse (*Eomenacanthus stramineum*) found principally in the region of the vent; the shaft louse (*Menopon gallinae*) located along the shafts of the feathers; the head louse (*Lipeurus heterographus*) found in the head and neck region, and the wing louse (*Lipeurus caponis*), detected along the primary feathers, are amongst the more important chicken lice, while the large turkey louse (*Goniodes meleagridis*) and the slender turkey louse (*Lipeurus gallipavonis*) are the most important turkey lice.

All species of lice complete their life cycle on the bird. As an example let us consider the common body louse. In this species eggs are deposited in large masses at the base of feathers, particularly in the region of the vent. These eggs, under favourable conditions, hatch in from five to seven days to produce nymphs. The nymphs, after several moults, give rise to adults. The whole process from egg to adult takes from two to three weeks.

Birds may be freed of lice by dusting with a suitable material. Sodium fluoride, sodium fluosilicate and 5% DDT powder are amongst the most effective and inexpensive materials known. Pinches of powder are applied to various parts of the bird's body, below the vent, in the thigh fluff, on the breast, under the wings, over the back and in the neck. One pound of powder used in this manner will treat about one hundred birds. Regardless of the

dusting material used, it is usually necessary to give a second treatment ten days after the first in order to kill any lice that may have hatched from eggs that were on the birds at the time of the first dusting.

Dipping of birds in warm water containing some material effective in destroying lice may be practised during warm weather. Sodium fluoride and sodium fluosilicate are the chief substances used in dipping solutions. One ounce of commercial powder or two-thirds of an ounce of chemically pure powder is added to each gallon of water. One pound of powder makes sufficient solution for dipping up to three hundred birds. Wettable DDT, at a concentration of 0.05%, may be used in the dipping solution in place of the aforementioned chemicals. In practice the birds are immersed, with the exception of the head, for twenty to thirty seconds. The feathers are ruffled so as to permit the dip to penetrate to the skin. Care should be taken to see that the dipped birds have ample time in which to dry before night. A second treatment ten days after the first is essential.

Mild greases such as lard and carbolated vaseline smeared on the top and back of the head may be used effectively in controlling head lice in young birds. Mercuric ointment, commonly known as blue ointment, diluted with vaseline 1:1 or a mixture of one part lard and one part sodium fluoride, have been found effective in combatting lice in older birds. A small quantity of the grease is applied just below the vent and under the wings. A second treatment is seldom required as the effect is lasting.

Fumigating with nicotine sulphate (Black Leaf Forty) is one of the easiest treatments yet discovered. The method consists of applying nicotine sulphate to the tops of the perches at the rate of eight ounces per 100 linear feet just before roosting time. The heat of the fowls' bodies causes the volatile nicotine sulphate to vaporize, the fumes penetrate the feathers of the birds and kill the lice. The roosts should be scraped free of organic material before the nicotine sulphate is applied. Care has to be taken to see that all birds roost and that no drafts are present, otherwise the treatment will not be successful. This treatment should only be used when the room temperature is over 55°F., and to be effective must be repeated in ten days time. Another method of using nicotine sulphate (one which costs less but involves more labour) is to apply it a drop at a time, by means of an oil can, directly to the bodies of the birds, under the wings and in the fluff around the vent. No second treatment is necessary in this case.

Spraying the roosts, dropping boards, adjacent walls and nests with 3.0% Malathion (Malathion Emulsifiable Liquid, American

Cyanamid Company), 0.5% chlordane (Chlordane Emulsion distributed by Commercial Chemicals) and 0.25% benzenehexachloride (sold under the trade name of "Lindane" by Canadian Industries Ltd.) have been found very effective in controlling chicken lice. Lindane may also be used as a roost paint for which purpose a 1.5% concentration of the active agent in oil is recommended. Malathion dust (5% Malathion) applied at the rate of one pound per twenty square feet of floor space is also highly effective as a treatment for lice.

The treatment of birds for lice should be followed by a thorough cleaning of the houses and premises. This is necessary, since the houses and runs are contaminated with lice and lice-egg laden feathers dropped from infested birds. If these are not destroyed, reinfestation will result.

For preventive measures for lice, see Mites.

**Mites.**

There are about twenty species of mites that infest domestic poultry. Of these, only four are sufficiently injurious to be of economic importance. They are the common chicken mite (*Dermanyssus gallinae*), the northern fowl mite (*Liponyssus silviarum*), the depluming mite (*Cnemidocoptes gallinae*), and the scaly leg mite (*Cnemidocoptes mutans*).

Mites are oval bodied creatures (see Fig. 57). Their bodies do not show head, thorax and abdominal divisions characteristic of lice. Their mouths are equipped for sucking blood.

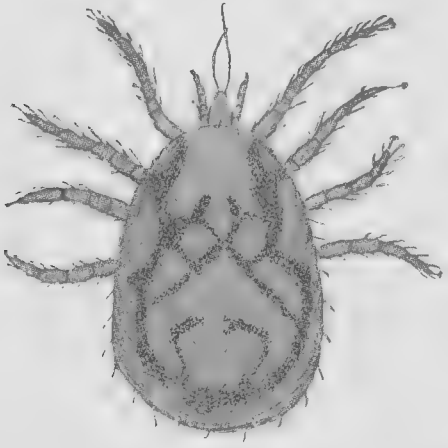


**Fig. 57—Common chicken mite.** (After Hutyra and Marck.)

Some species of mites complete their life cycle on the bird while others do not. Generally speaking, the time needed to complete the life cycle is short; the average time required from egg to adult, under favourable conditions, being seven or eight days.

*Common Chicken Mite.*—The common chicken mite, also referred to as the red mite, because of its red appearance when gorged with blood, is gray in colour. This mite does not stay on the fowl all the time. It is found on the bird only at night (in cases where infestation is exceedingly heavy it may be found on the bird during the day). During the day it hides in cracks and crevices. Its presence may be determined by examining the ends and undersides of the roosts at the points of support, and by careful examination of any cracks or crevices in the roosts or roost supports. "Salt-and-pepper markings" (the excrement and sloughed-off skin of mites) under or on the roosts are also an indication of the presence of red mites. Examining a representative group of birds after dark is another way in which the presence or absence of this mite may be determined.

If evidence of infestation is seen, measures of control should be taken at once. The house and house equipment should be cleaned. Roosts, dropping boards, adjacent walls and nest boxes should be thoroughly sprayed with a 5% water solution of wettable DDT, a saturated coal oil solution of DDT, a 4.0% nicotine sulphate solution or a 1.0% Malathion solution. A second treatment, after a lapse of ten days, is advised.

*Northern Fowl Mite.*—This parasite somewhat resembles the common chicken mite in appearance. It differs, however, in habit, in that it completes its life cycle on the bird and accordingly its presence or absence may be ascertained by examining the fowl at any time. Where infestation is heavy, large numbers of the mites may be seen on the surface of the feathers. Northern fowl mites tend to congregate in certain areas for feeding. Often the injury inflicted in these areas is so severe that large scabs form.

There are several satisfactory methods of treating for this mite, namely, fumigating with nicotine sulphate (Black Leaf Forty), spraying with 0.5% Malathion (American Cyanamid Company) solution at the rate of one gallon of spray per hundred birds, dusting with 1½% Malathion dust or with 10% Neotran (Dow Chemical Co.), greasing with a mixture of one part napthalene to two parts vaseline, or, during warm weather, by dipping the birds in a solution of two ounces of sulphur plus one ounce of soap in one gallon of warm water. Fumigation with nicotine sulphate is carried out in a manner similar to that outlined for lice (see Lice), except that three applications at intervals of three days are necessary in

this instance. Nicotine sulphate may also be applied as a 4% spray to the interior of the house and to the equipment. In this case two applications, three days apart, are recommended. The method followed in greasing or dipping is the same as that described under Lice.

*Scaly Leg Mite.*—This mite burrows under the scales of the shanks, causing the condition known as scaly-leg (see Fig. 58). The scaly leg mite completes its life cycle on the bird.

**Fig. 58—A bad case of scaly-leg. This condition is caused by the scaly leg mite.** (After Iowa State College.)

Treatment may be effected by applying to the shanks an ointment of 1 part of oil of caraway to 5 parts of vaseline or 2 parts carbolic acid to 98 parts of vaseline; or by dipping the shanks in petroleum, crankcase oil, kerosene, a mixture of 1 part kerosene to 2 parts raw linseed oil; or in a mixture of 1 part crude petroleum to 1 part raw linseed oil. In connection with the ointment treatment, before applying the salve it is advisable to soak the shanks in warm soap suds until the scales are loose. Two applications at three or four day intervals are necessary. Where the dipping-of-shanks method of treatment is followed, care should be taken to see that the dip does not get on the skin of the bird, otherwise burning will result.

*Depluming Mite.*—The depluming mite burrows into the skin at the base of the feathers, causing an intense itching which causes the bird to pick out its feathers. As a result, large areas on the bird may be devoid of feathers. Absence of feathers from the backs and/or abdomens of birds should not, however, be assumed

to be caused by depluming mite until the possibility of the presence of the cannibalistic habit of feather picking has been ruled out (see Cannibalism).

Treatment for depluming mite consists of applying to the affected areas either oil of caraway ointment (see above) or sulphur ointment (1 part flowers of sulphur to 4 parts lard or vaseline), or, during warm weather, by dipping in a solution of two ounces of sulphur plus one ounce of soap to one gallon of warm water. At least two applications of ointment should be given. Where dipping is resorted to, the birds must be thoroughly soaked for the treatment to be effective.

While definite methods of treatment for lice and mites have been outlined, it should be realized that cleanliness, proper housing and vigilance constitute the most effective means of controlling external parasites. Houses should be thoroughly cleaned, whitewashed and sprayed with disinfectant at least once a year, better twice; litter and nesting material should be changed at least every two weeks and houses and equipment should be so constructed as to provide for a minimum of cracks in which mites might hide and breed. In addition the birds and the houses should be examined regularly for the presence of external parasites. As soon as their presence is detected control measures should be instituted.

## INTERNAL PARASITES

There are two main types of worms that affect poultry, namely, roundworms (*Nematodes*) and tapeworms (*Cestodes*). Roundworms are readily distinguished from tapeworms by morphology, the former being round and unsegmented, the latter flat and segmented. Both types are usually white in colour and vary in length according to species. The large intestinal roundworm (*Ascaridia lineata*), the caecal worm (*Heterakis gallinae*), and the gapeworm (*Syngamus trachea*) are among the more important roundworms that infest the fowl. Several species of tapeworm are known to infest the fowl.

**Large Intestinal Roundworm.**

This worm (see Fig. 59) inhabits the small intestine. It is white in colour and varies in length from one to three inches. Its life cycle is direct (see Fig. 60). Non-infective eggs (non-infective to the fowl) are laid in the intestine by the adult female worm. These eggs pass out of the body along with the faeces. Under suitable conditions of moisture and heat, these non-infective eggs undergo development (embryonation) to give rise to infective eggs. Upon being ingested by a fowl these infective eggs pass down

into the small intestine where they liberate larval worms. The larval worms so released enter the intestinal wall where they undergo further development to produce normal worms which, after passing out into the intestine, grow to maturity and lay their complement of non-infective eggs.

**Fig. 59.—Section of intestine showing large intestinal roundworms protruding.** (After Bradshaw.)

Old birds are less susceptible to large intestinal roundworm infestation than young birds. This difference is because of the development of a so-called "age immunity" as fowls grow older. Birds on an inadequate diet are more susceptible to intestinal roundworm infestation than are birds receiving a properly balanced diet. Birds on restricted feed appear to be more subject to worm infestation than those on full feed.

The principal symptoms of large intestinal roundworm infestation in growing fowls are: paleness of head and shanks, loss of appetite, stunted growth, emaciation, inactivity, and droopiness. In advanced cases lameness or paralysis is often noted. In mature fowls decreased egg production, loss of weight and general unthrift should be regarded as indications of roundworm infestation.

While the above symptoms are suggestive of the presence of roundworms, they do not constitute conclusive evidence. The presence or absence of roundworm infestation is best ascertained by examining the intestinal contents of suspected birds.

On farms where infestation prevailed in the previous season, ground over which the birds ranged constitutes the main reservoir of infection. This is because worm eggs are able to persist in the soil in a viable state from one season to the next. On farms where current infestation exists, the dropping boards (especially if not cleaned daily), litter, contaminated feed and water, and the ground

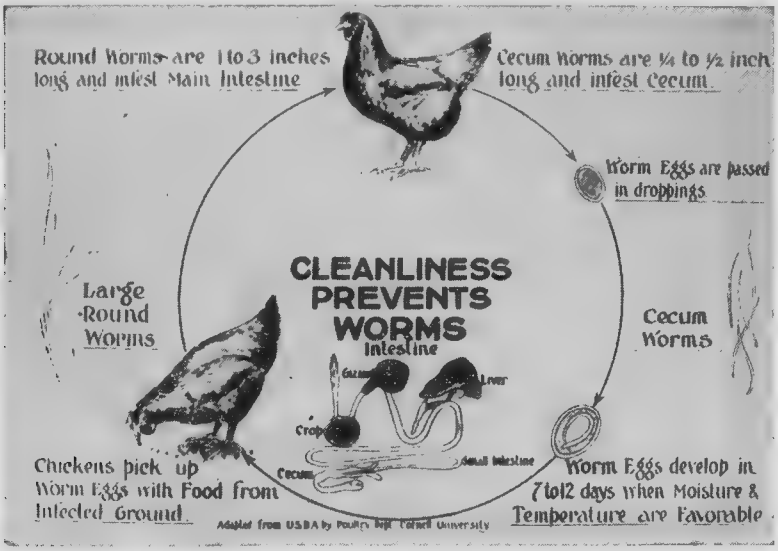

Round Worms are 1 to 3 inches long and infest Main Intestine
Cecum Worms are ¼ to ½ inch long and infest Cecum
Worm Eggs are passed in droppings
CLEANLINESS PREVENTS WORMS
Intestine
Large Round Worms
Cecum Worms
Chickens pick up Worm Eggs with Food from Infected Ground
Worm Eggs develop in 7 to 12 days when Moisture & Temperature are Favorable
Crop
Liver
Cecum
Small Intestine
Adapted from USDA by Poultry Dept. Cornell University

**Fig. 60—Life cycle of large intestinal roundworm and of caecal worm.**
(After Cornell University.)

over which the birds range, comprise the principal sources from which healthy fowls contract infection.

Worm infestation has a decided detrimental effect on vigour. Worm infested birds are much more susceptible to disease than healthy birds. Mortality from large intestinal roundworm infestation varies greatly. In general, losses in young stock are more serious than in adult birds.

Trouble from large intestinal roundworms may be avoided by observing:

(1) Sanitation on the range.

(a) Using poultry ranges one year in two; or better, one year in three, to ensure freedom from worm eggs.

(b) Avoiding damp spots on the range. Such spots provide an ideal environment for the embryonation of worm eggs. All low damp spots should be filled in or fenced off. Unused drinking water should be disposed of in such a manner as not to leave puddles on the range. Water fountains should be placed up on wire stands to prevent birds having access to the damp ground usually present about them.

(2) Sanitation in the brooder house.

(a) Cleaning out the brooder house thoroughly before use to destroy worm eggs that may be present. One pound

of lye to 5 gallons water should be used for disinfecting the floor.

(b) Using wire under the roosts to prevent the birds from coming in contact with their faeces.

(c) Cleaning the litter and droppings out at regular intervals to lessen the chances of worm-spread.

(3) Sanitation in the laying house.

(a) Cleaning out the laying house thoroughly prior to housing pullets to destroy worm eggs that may be present. See recommendations above for disinfection.

(b) Cleaning the dropping-board daily or using wire netting under the roosts to prevent the birds from coming in contact with their faeces.

(c) Cleaning the litter out at regular intervals to lessen the chances of worm-spread.

(4) Sanitation in feeding.

(a) Protecting feed and water against contamination with droppings to avoid spreading infection. All feeders and waterers should be provided with reels to prevent birds getting into them. Landing boards in front of the feeders and waterers should be sufficiently far away from same to prevent birds from soiling feed and water with droppings.

(5) Sanitation in management.

(a) Rearing young stock separate from old stock. The latter frequently harbour worms, hence must be considered as a potential source of infestation.

(b) Taking precaution against mechanical transmission of infection from old stock to the young stock.

(c) Being careful not to bring infection on the farm. Imported birds should be treated for worms if infestation is suspected. Visitors should not be permitted to enter the poultry houses, yards and ranges.

Treatment of birds infested with roundworms may be effected by administering a vermifuge. Several good vermifuges are obtainable from poultry supply houses, Iodine Vermicide, (Merck and Co. Ltd.); PN Powder or Tablets (Lederle Laboratories Inc.); Rotacaps (Dr. Salsbury's Laboratories). Piperazine compounds have also been shown to be highly effective in eliminating roundworms. For details of administration of the above referred vermifuges the reader is referred to the manufacturers' instructions. Two chemicals

that are recommended widely for treatment of roundworms are tetrachlorethylene and carbon tetrachloride. One cubic centimeter of either constitutes a dose for an adult bird, while 0.5 c.c is sufficient for a growing bird. The following procedure is recommended when either of these two chemicals is used:

(1) Treat affected birds in the morning before they are permitted to feed.

(2) Administer the proper amount of chemical into the crop of each bird, either in capsule form or by means of a glass or rubber tube.

(3) Give Epsom salts in the drinking water at the rate of one pound per hundred birds.

(4) Supply regular feed two hours after treatment.

(5) Confine birds to a house for three days following vermifuge administration.

(6) Clean the house out thoroughly on the third day, using lye water (1 pound of lye to 5 gallons of water) for disinfecting.

Following the treatment every effort should be made to avoid reinfestation. Under no circumstances should the treated birds be permitted to range over ground on which they previously ranged. Young stock in colony houses should be moved to a clean piece of range, while old birds in permanent houses should be confined until such time as their yards have been cleaned up.

Worm-egg infested ground is best cleaned up by ploughing and cultivating frequently. One summer's working and a year's rest will clean up a worm-egg-infested range.

**Caecal Worm.**

These small white worms (¼ to ¾ inches long) inhabit the blind guts or caeca of the birds. The life cycle of this worm is similar to that of the large intestinal roundworm (see Fig. 60).

Most classes of poultry are susceptible to caecal worm infestation. Birds in a state of malnutrition (e.g. deficient in vitamin A) are more susceptible to caecal worms than those in a state of proper nutrition.

The symptoms, sources of infection, and effects of caecal worm infestation are similar to those referred to under Large Intestinal Roundworm. Post-mortem examination of infested birds reveals large numbers of worms (see Fig. 61) toward the tips of the caeca.

It should be pointed out that the caecal worm is dangerous in the poultry yard, not only because it is capable of infesting birds and causing trouble, but also because of the part it plays in the transmission of blackhead (see Blackhead).

**Fig. 61—The caecal worm from blind gut of a chicken.**
(After Biely, Lloyd and Moynihan.)

Preventive measures for caecal worms are the same as for large intestinal roundworms (see Large Intestinal Roundworm).

Treatment of infested birds is somewhat difficult as the caecal worm inhabits a rather inaccessible part of the intestine. Administering 2%, by weight, of freshly pulverized tobacco (containing not less than 1½% nicotine) in the mash for four to six weeks, or giving birds over three pounds in weight a rectal injection of 2/3 of a fluid ounce (birds under three pounds in weight 1/3 of a fluid ounce) of a mixture of one fluid drachm of oil of chenopodium (oil of American wormseed) and 6 fluid ounces of vegetable oil, are two methods of treatment for caecal worms.

In connection with the former treatment, the mash and tobacco dust mixture should be made up frequently in small amounts rather than in one large lot. This is advisable because the tobacco loses its nicotine rapidly on exposure to air. In the latter treatment, an enema syringe with a small blunt nozzle is used for injecting the oil of chenopodium-vegetable oil mixture.

A third method for treating for caecal worms and one that is highly recommended consists of administering the drug phenothiazine at the rate of one pound per thousand chickens or growing turkeys, or at the rate of two pounds per thousand adult turkeys. The drug may be given either in capsule form or may be fed in the mash.

Regardless of the treatment used, reinfestation must be avoided by following a plan similar to that given under Large Intestinal Roundworm.

**Gapeworm.**

Gapeworms are small Y-shaped worms of reddish brown colour (see Fig. 62). The short part of the "Y" is the male and the long part the female. These worms attach themselves to the mucous membrane of the trachea (windpipe) and suck their food (blood) therefrom. The life cycle of this worm is similar to that of the large intestinal roundworm (see Fig. 60).

**Fig. 62—The gapeworm. Open trachea showing gapeworms attached.**
(After Moynihan.)

Both chickens and turkeys are highly susceptible to gapeworm infestation. Heavy losses may be experienced where chickens, especially young chickens, are infested with these worms. Turkeys however, may harbour numerous worms without showing adverse effects.

The chief symptoms noted in gapeworm infestation are gasping for breath and inactivity. Death occurs through suffocation. Post-mortem examination reveals the presence of reddish brown Y-shaped worms in the windpipe.

Sources of infection for gapeworms are similar to those referred to under Large Intestinal Roundworm.

Preventive measures for gapeworms are the same as for large intestinal roundworms (see Large Intestinal Roundworm); with one addition, namely, that chickens should be reared separate from turkeys. This is advisable since turkeys are potential carriers of gapeworms.

A treatment that has proven successful in removing gapeworms consists of pipetting 1/3 to 3 minims of a mixture of equal parts garlic oil or allyl sulphide and linseed oil into the windpipe of affected birds. The drug causes the parasites to release their hold and the birds cough them up. An alternative treatment that is highly recommended consists of exposing affected birds to air containing barium antimonyl tartrate dust. The birds are exposed to the chemical in a closed container, the size of the dose being determined by the cubic capacity of the container used. One ounce of barium antimonyl tartrate is sufficient for a box having a capacity of 8 cubic feet. Infested birds are placed in the box in sufficient numbers to occupy about two-thirds of the floor space. The barium antimonyl tartrate is introduced with a dust gun into the box through a hole in the top. The treatment should take about 15 minutes.

Reinfestation should be guarded against by following a plan similar to that outlined under Large Intestinal Roundworm.

**Tapeworms.**

As previously stated, tapeworms are flat, segmented worms of white colour (see Fig. 63). The species that infest poultry vary in size from those that cannot be seen by the unaided eye to worms nine inches in length. The life cycle of the tapeworm is indirect, i.e., one phase of it is spent inside the body of a secondary or intermediate host (see Fig. 64). Non-infective eggs (non-infective to the fowl) are passed in the droppings of infested birds. Then these non-infective eggs are ingested by suitable secondary or intermediate hosts (fly larvae, flies, snails, beetles, grasshoppers, earthworms, etc.). While inside the intermediate host, they undergo

development to produce infective larvae (*cysticerci*). Next the intermediate host containing the infective larva is eaten by the fowl. During the course of digestion, the head (scolex) within the cyst is released and attaches itself to the wall of the upper intestine by means of hooks and suckers. During the course of the development that ensues, new segments (proglottids) are formed in the region

**Fig. 63—Tapeworms attached to the lining of the small intestine.** (After Beach.)

behind the head (strobilus). This development continues until the tapeworm has grown its characteristic length, at which time the oldest proglottids (which are filled with non-infective eggs) detach, to be later passed out of the body of the fowl with the faeces.

Chickens and turkeys are both susceptible to tapeworms.

The symptoms of tapeworm infestation are similar to those enumerated under Large Intestinal Roundworm. In heavy tapeworm infestations segments can often be observed in the droppings.

Post-mortem examination of affected birds reveals an inflamed upper intestine and the presence in the intestine of segmented, flat, white worms. Of course, where microscopic species of tapeworms are concerned, it is impossible to see the worms without the aid of a microscope. One species of tapeworm (*Raillietina echinobothrida*) produces small nodules in the walls of the intestine. These nodules should not be mistaken for tuberculosis nodules.

The damage done by a few tapeworms in mature birds is not very great, but a heavy infestation, especially in young birds may have disastrous results. It should, however, be pointed out that tapeworm infestation, regardless of the severity of the infestation, like roundworm infestation tends to undermine the bird's vitality and to predispose to secondary infection. This in itself constitutes sufficient reason for taking precautions to control the tapeworm menace.

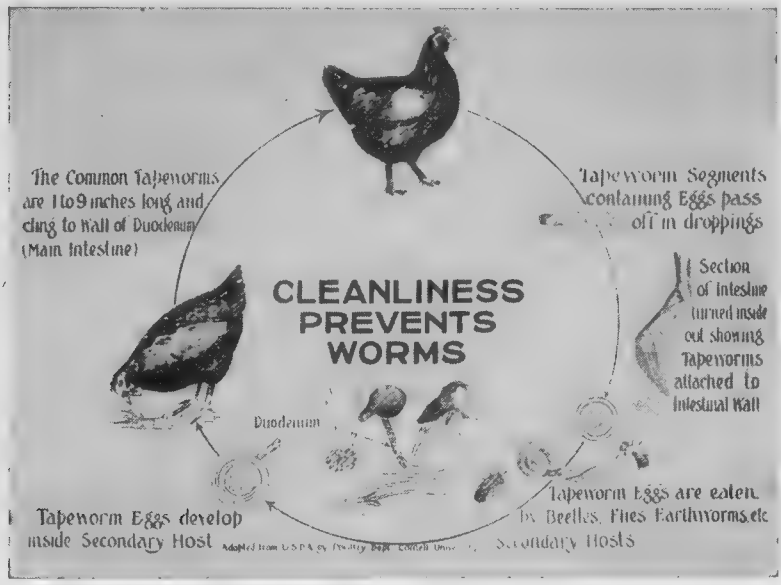

The Common Tapeworms are 1 to 9 inches long and cling to Wall of Duodenum (Main Intestine)
Tapeworm Segments containing Eggs pass off in droppings
Section of Intestine turned inside out showing Tapeworms attached to Intestinal Wall
CLEANLINESS PREVENTS WORMS
Duodenum
Tapeworm Eggs develop inside Secondary Host
Tapeworm Eggs are eaten by Beetles, Flies Earthworms etc
Secondary Hosts

**Fig. 64—Life cycle of poultry tapeworm.** (After Cornell University.)

The preventive measures listed under Large Intestinal Roundworm are applicable to tapeworms. In addition any measures that will lead to a reduction in the number of intermediate hosts on the poultry farm (such as the spraying with Malathion or DDT around dropping boards and manure pits to kill possible intermediate hosts and the removal of all old boards and debris, etc., which provide a breeding place for intermediate hosts) or to prevention of intermediate hosts from coming in contact with worm-egg-infected droppings (such as by frequent removal of droppings and litter) will prove of value in combatting the tapeworm problem.

Treatment of birds infested with tapeworms is very difficult. No chemical is known which, when administered to an infested bird, will cause the expulsion of all tapeworms intact. Vermifuges are known that will destroy all the segments, but none is known that

will destroy all the heads. The latter remain attached to the intestinal wall and develop a new lot of segments. Hence, administering a vermifuge for tapeworms merely results in a temporary cessation of tapeworm activity.

Iodine Vermicide (Merck and Co. Ltd.) and Cloro-caps (Dr. Salsbury's Laboratories) are considered amongst the best tapeworm vermifuges available. For details of administration the reader is referred to the manufacturers' instructions. Recently, di-n-butyl tin dilaurate administered at the rate of 75 to 125 mg. per bird or 500 mg. per kilogram of ration for 2 to 6 days has been found to be highly effective against tapeworms. Single doses of as little as 16 mg. of di-n-butyl tin (DBT) oxide, 37.5 mg. DBT maleate or dibenzyl tin dichloride, or 75 mg. of DBT sulphide have also been found effective in the treatment of birds infested with tapeworms.

**The Province of Alberta provides a free poultry disease diagnostic service. Farmers who wish to use this service should take or ship their ailing or dead specimens to the Poultry Pathologist, Provincial Veterinary Laboratory, Edmonton, Alberta. A letter addressed to the Poultry Pathologist, Provincial Veterinary Laboratory, Edmonton, giving details on case history and symptoms should accompany or follow the shipment.**